Cardiology

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وَقُلِ اعْمَلُواْ فَسَيَرَى اللّهُ عَمَلَكُمْ وَرَسُولُهُ وَالْمُؤْمِنُونَ وَسَتُرَدُّونَ إِلَى عَالِمِ الْغَيْبِ وَالشَّهَادَةِ

فَيُنَبِّئُكُم بِمَا كُنتُمْ تَعْمَلُون ۝ (التوبة-105)

Say, Work righteousness; GOD will see your work, and so will His messenger and the believers. Ultimately, you will be returned to the Knower of all secrets and declarations, then He will inform you of everything you had done.

Al-Tawba Verse No: 105

Dedication

To the soul of my mother

Contents

INTRODUCTION

The epidemic of cardiovascular diseases (CVDs) is accelerating globally over all regions & social classes. This is reflected in the high burden as well as the estimated escalation of those burdens over the next 2 decades. CVD contributes to a large proportion of morbidity & mortality. As many as 39% of patient (pts) with (é) acute rheumatic fever may develop varying degrees of pancarditis é associated valve insufficiency, heart failure (HF), pericarditis & even death. A number of reasons underlie the expected rise in CVDs as:-

- An overall ⇧ in the population.
- Improved life expectancy is leading to more person living to the middle age & beyond, which (ẃ)⇧ the risk of CVDs.
- Lifestyle transition; ⇧ urbanization, industrialization, globalization & change in nutritional habit.
- Past or current nutrition deprivation in utero & early childhood may affect cardiovascular health trend.
- Lack of weight gain in the 1st year (yr) of life & low birth weight (LBW) in spite of maternal weight gain have been linked to coronary disease in adult life.

The causes of CVDs in developing countries include:-

- Chronic rheumatic heart disease.
- Hypertension & ischemic heart disease (IHD).
- Cardiomyopathies.
- Congenital disease.

There are indicators of ⇧ prevalence of IHD due to the existence of risk factors in some segment of population these includes:-

- Hypertension. - Hypercholesterolemia. - DM. - Obesity. - Smoking.

RHEUMATIC FEVER

RF cause chronic progressive damage to the heart & its valves. The association between sore throat & RF was not made until 1880. The dramatic decline in the incidence of RF in the developed world is thought to be largely owing to antibiotic Rx of streptococcal infection, though it stated to decline before the era of antibiotic, probably due improvement of socioeconomic status. It thought that 40-60% of pts é acute rheumatic fever (ARF) will develop rheumatic heart disease (RHD). The commonest valves affected are the mitral & aortic, in that order. However all four valves can be affected.

Epidemiology

Even though the overall incidence of RF & RHD in developed countries has sharply declined, RF is the commonest cause of heart disease (HD) in the developing countries. In those countries >50% of HD is accounted for RHD. Even in developing countries, the prevalence of RF & RHD varies between rural & urban areas. As many as 39% of pts é ARF may develop varying degrees of pancarditis é associated valve insufficiency, HF, pericarditis & even death. Worldwide, there are >15 million cases of RHD é 282.000 new cases & 233.000 deaths from this disease each year. The incidence in developed countries 0.5-3/100.000 & in developing countries is as high as 100-200/100.000. The overall mean incidence of RF worldwide: 5-50/ 100.000. The age 5-15 yrs are most susceptible & it is rare in age < 3 yrs. The girls are affected 〉 Boys. The incidence more during fall, winter & early spring. The high attack rate of group A streptococcal pharyngitis in families, institutions & military recruits is the result of contact among susceptible persons living closely enough to ensure droplet transmission. The risk factors for RF include low socioeconomic status é associated overcrowded living condition. However, RF is not associated é streptococcal skin infection (pyoderma)?.

Pathophysiology

ARF is a sequel of a previous group A streptococcal (GAS) infection, usually of the URT. One ß-streptococcal serotype (e.g. M types 3, 5, 18, 19, 24) is linked directly to ARF. Rheumatogenicity of GAS is important factor as not all GAS pharyngitis is associated é development of RF. RF follows Lancefield β hemolytic streptococcus pharyngitis within the interval of 2-3 wks. The mechanism is elusive, but the followings are proposed ones:-

- Dysfunction of the immune response.
- Antigenic mimicry: similarity between the carbohydrate moiety of GAS & glycop-roteins of heart valve. Molecular similarity between some streptococcal antigens & sarcolemma or other moiety of human myocardial cells.
- Several host related factors have been identified to have operated in relation to specific genetic function & difference in the immune response of individuals.
- The disease characterized by an exudative & proliferative inflammatory lesion of the connective tissue especially that of the heart, joints, bl. vessels & subcutaneous tissues.

Clinical manifestation

ARF is associated é 2 distinct patterns of presentation:-

- First pattern of presentation is sudden onset, typically begins as polyarthritis 2-6 wks after streptococcal pharyngitis & usually characterized by fever & toxicity.
- Second pattern is insidious or subclinical & the initial abnormality is mild cardities. The age at onset influence the order of clinical picture, younger children tend to develop cardities first, older pt tend to develop arthritis first.

Diagnosis

RF is mainly a clinical diagnosis. No single diagnostic sign or specific laboratory test available for diagnosis. 50% of cases of RHD **don`t remember** having ARF. The diagnosis requi-

res high index of suspicion. Jones criteria developed by the American Heart Association is used to make the diagnosis include the following;

Major criteria	Minor criteria
Pancarditis (pericarditis, endocarditis, myocarditis)	Fever
Polyarthritis	Arthralgia
Sydenham Chorea	Prolonged PR interval
Subcutaneous Nodules	Increased ESR or CRP*
Erythema marginatum	Leukocytosis

2 major or 1 major + 2 mino
must be present for diagnosis of Rheumatic Fever

Diagnosis of ARF *requires 2 major or 1 major + 2 minor criteria*

1) Cardities (pancarditis here): occurs in as many as 50% of pts & may manifest as;

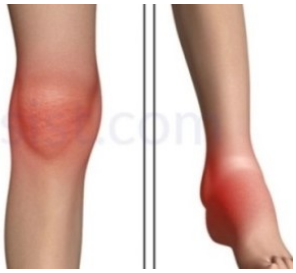
(a) New murmur. (b) Cardiomegaly. (c) CHF. (d) Pericarditis é/éout a pericardial rub & resolve éout constriction. (e) Valvular disease: mitral & aortic valves are commonly affected. Healing of rheumatic valvulitis will lead to fibrous thickening & adhesion, resulting in progressive valvular damage. But, about 80% of mild valvulitis would resolve. There is a risk of developing endocarditis on a damaged valve.

2) Migratory polyarthritis: occurs in 75% of cases, fleeting arthritis é all signs of inflammation on the joint, the larger joints are mainly affected, lasts for 2-3 days then migrate to other joints. Arthritis do not progress to chronic disease. Involves many joints at a time.

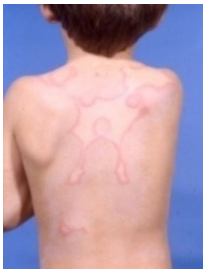
3) SC nodules: occur in 10% of pts & are edematous fragmented collagen fibers. They are firm painless nodules on the extensor surfaces of wrists, elbows & knees, seen over bony prominence or in heart (Aschoff nodules) & associated é cardities.

4) Erythema marginatum: occurs in 5% of cases. The rash is serpiginous & long lasting, occur in the trunk or lower limbs, clear from center & spread peripherally.

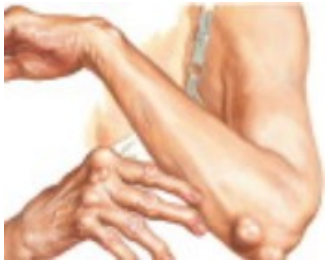
5) Sydenham's chorea: "St Vitus 'dance", is characteristic movement disorder occurs in 5-10% of cases. Consists of rapid purposeless movements of face & upper extremities. Onset may be delayed for several months to years & may cease when the pt is asleep & seen mainly in girls.



Typical appearance of Arthritis



Erythema Marginatum



Subcutaneous Nodules

Laboratory studies

No specific confirmatory laboratory tests exist. However, several laboratory findings indicate continuing rheumatic inflammation. Some are part of the Jones minor criteria.

- ASOT is +ve in 80% of cases.
- Anti DNAase β & Anti hyaluronidase +ve in 95% of cases.
- Isolate GAS via throat culture ẃ has 20-40% yield.
- ESR &CRP. Leucocytosis may be seen.
- Anemia from suppression of erythropoiesis.
- Prolonged P-R interval in 25% of all cases but is neither specific nor diagnostic.

Treatment

1. Treat group A streptococcal infection: regardless of organism detection, all pts é ARF should be given appropriate antibiotic. Penicillin V 250.000 u/5 ml or Cliacil 1.200.000 u/tab., at a dose of 100.000 u/Kg/day ÷ 4 for 10 days. Or Benzathin Penicillin 1.2 million u IM as single dose, or Erythromycin 500 mg PO QID for 10 day (for Penicillin allergic pt).

2. Therapy for manifestation of ARF.

Arthritis: ASA is given at dose 2 gm 4 times/D for 4-6 wks, at a dose of 100 mg/ Kg/D ÷ 4,

maximum dose 10 gm/day + antacid. ⇩ the dose according to the results of ESR, or after 1 wk to 50% & continue for 1 month. No indication for steroids.

Cardities: severe cardities é CHF should be treated é prednisolone 60-80mg/day, I mg/ Kg /day÷4 for 4 wks, to be tapered as pt improves. Start ASA during tapering phase to be given for 4-6 wks, but both have no influence on development of valvular heart disease.

CHF: conventional Rx such as Digoxin & Diuretics.

Sydenham's chorea: the majority of cases are self-limiting, but in symptomatic pt Benzodiazepines (Diazepam) or Phenothiazines (Haloperidol) may be helpful in controlling symptoms.

3. Complete rest in bed 10 days.

4. Administer secondary prophylaxis: indicated for all pts é RF. Benzathin Penicillin is the 1st choice for better compliance & longer prevention, 1.2 million IU IM/4 wks, but if there is high risk of recurrence, it can be given every 3 wks, or Penicillin V syrup (250 mg twice/D), or Sulfadiazine (1 gm/D), or Erythromycin for those allergic to Penicillin. In pt é an established RHD, it is advisable to be given life long.

Epidemiology	• Peak incidence age 5-15 • Twice as common in girls	
Clinical features	Major	•Joints (migratory arthritis). • Carditis. •Nodules (subcutaneous). •Erythema Marginatum. •Sydenham chorea.
	Minor	•Fever. •Arthralgia. • Elevated ESR./ C- reactive protein. •Prolonged PR interval.
Late seqelae	Mitral regurgitation/stenosis	
Prevention	Penicillin for group A strept (pyogenes) pharyngitis	

CONGESTIVE HEART FAILURE

Heart failure is a clinical syndrome characterized by inadequate systemic perfusion to meet the body's metabolic demands as a result of abnormalities of cardiac structure or function. This may be further subdivided into systolic or diastolic HF:

- Systolic HF: there is reduced cardiac contractility.
- Diastolic HF: there is impaired cardiac relaxation & abnormal ventricular filling.

In HF the body, sensing inadequate organ perfusion, activates multiple systemic neurohormonal pathways ẃ compensate initially by redistributing blood flow to vital organs, but later exacerbate the pt's symptoms & lead to deterioration.

Etiology

HF is the most common reason for hospitalization in adults >65 yrs old. About 30% of pts experiencing myocardial infarction (MI) also develop HF. The most common cause of HF is systemic hypertension (60-70% of pts). The following are some underlying causes:-

- ⇩ ***Of the contractile function of the heart:*** as é valvular or coronary heart disease (myocardial ischemia, MI) or myocardial disease (cardiomyopathy & myocarditis).
- ⇧ ***Of the afterload:*** acute systemic hypertension.
- ***Abnormalities in the preload:*** as é excessive or reduced preload.
- ⇩ ***Compliance***: as é constrictive pericarditis or restrictive cardiomyopathy.

Preload: is the end diastolic pressure that stretches the right (Rt) or left (Lf) ventricle to its greatest geometric dimensions under variable physiologic demand. It depends upon venous return & compliance.

Afterload: is the force needed to eject blood into circulation. The pressure in ventricles must be more than the systemic & pulm pressures to open the aortic & pulm valves.

The afterload ⇧ é valvular diseases.

Precipitating factors for CHF

These are relatively acute disturbances that place an additional load on a myocardium that is chronically & excessively burdened. In compensated state pt is asymptomatic; however as pt have little additional reserve, he become symptomatic in the presence of these ppt factors. Knowing the precipitating factors is important because most of the time they are treatable & the cardiac function improves when these precipitating factors are treated or avoided. The most important precipitating factors may be represented é the mnemonic, "HEART FAILES";

•H Hypertension (systemic). •E Endocarditis (infections) •A Anaemia. •R RF & myocarditis. •T Thyrotoxicosis & pregnancy. •F Fever (infection). •A Arrhythmia. •I Infarction. •L Lung infection. •E Embolism (pulmonary). •S Stress (Emotional, Physical, Environmental, Dietary & Fluid excess).

Pathophysiology

In Lf ventricular systolic dysfunction, regardless of the etiology, the COP is low & pulmonary pressures are high, leading to pulmonary congestion. As a result, a series of adaptive mechanisms are activated. Initially, as direct result of inadequate COP & systemic perfusion, the body activates several neurohormonal pathways in order to ⇧ the circulating blood volume & é continuous neurohormonal stimulation, the LV undergoes remodelling é LV dilatation & hypertrophy, such that stroke volume is ⇧ éout an actual ⇧ in ejection fraction (EF). This is achieved by myocyte hypertrophy & elongation. However, LV chamber dilatation causes ⇧ wall tension, worsens subendocardial myocardial perfusion & may provoke ischemia in pt é coronary Atherosclerosis. Furthermore, LV chamber dilatation may cause separation of the mitral leaflets & mitral regurgitation é worsening of pulmonary congestion. Enhanced neurohormonal stimulation of the myocardium also cause

apoptosis or programmed cell death, lead- ing to worsening of ventricular contractility.

Clinical manifestations

- **Progressive dyspnea:** ẃ initially occurs é exertion & later occurs at rest. Found to be the most sensitive complaint, yet the specificity for dyspnea is **<** 60%,
- **Orthopnea & paroxysmal nocturnal dyspnea:** specific symptoms, sensitivity is 20-30%.
- **Cough:** productive of pink, frothy sputum is highly suggestive of CHF.
- **Peripheral edema & ascites.**
- **Nonspecific complaints:** easy fatigability, light headiness, malaise, anxiety.
- **Past medical history**: of RF, Alcohol use, Hypertension, Angina, previous history of MI & +ve family history.

Physical findings

- ***Tachycardia, Tachypnea, signs of Res.Distress*** (use of accessory muscles of respiration).
- ***Jugular venous distension*** frequently present & engorged neck veins.
- ***Pulses alternans:*** weak & strong pulse indicative of depressed LV function.
- ***Wheezing or rales:*** may heard, bilateral basal dullness may elicited.
- ***Apical impulse frequently displaced:*** laterally.
- ***Cardiac auscultation:*** may reveal aortic or mitral valvular abnormalities, S3 or S4.
- ***Skin may be diaphoretic or cold, grey & cyanotic peripheral oedema*** may noticed.

Grades of CHF

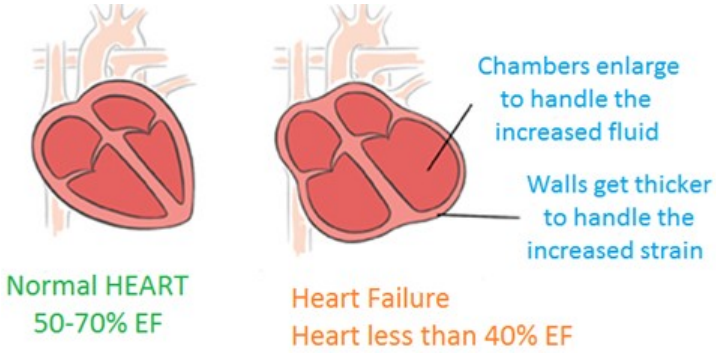
Grade	Symptoms
I	No symptom limitation é ordinary physical activity.
II	Ordinary physical activity somewhat limited by dyspnoea (i.e. long distance walking, climbing 2 flights of stairs).
III	Exercise limited by dyspnoea at mild workloads (i.e. short distance walking, climbing one flight of stairs).
IV	Dyspnoea at rest or é very little exertion.

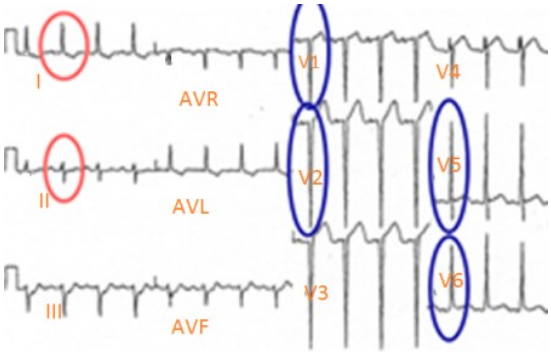
Diagnostic workup

- ***CXR***: cardiomegaly, pulmonary oedema & pleural effusion.
- ***ECHO***: Key indicator for diagnosing. The EF is the % of blood that is pumped out of the heart during each beat. EF normally 50-70%, ⇩ é HF to ‹40%. ECHO also help to detect valvular abnomalies, ventricular dysfunction, cardiac tamponade, pericardial constriction & pulmonary embolus.
- ***ECG***:for diagnosing concomitant cardiac ischemia, prior MI, cardiac dysrhythmias & LVH.

☆ **To Diagnose *LVH*** you can use the following criteria*:-*

R wave in V_5 or V_6 + S wave in V_1 or V_2 are ›35 mm & the R wave in AVL ›13 mm.

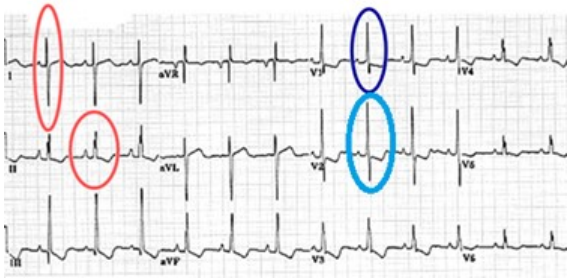




The above ECG strip shows left axis deviation R wave is +ve in lead I & -ve in lead II, III + tall R waves in V_5,V_6. Deep S waves in V_1,V_2 . The deep S waves seen in leads over the Rt ventricle are created because the heart is depolarizing (away from V_1, V_2).

☆ **To Diagnose *RVH*** you can use the following criteria:-

Rt axis deviation (R wave is - ve in lead I + ve in lead III + R wave in V_1 ›7 mm tall.



The above ECG shows Rt axis deviation + tall R waves ›7 mm tall in V_1, V_2 = RVH.

☆ **To Diagnose *Left Atrial Enlargement,*** you can use the following criteria:-

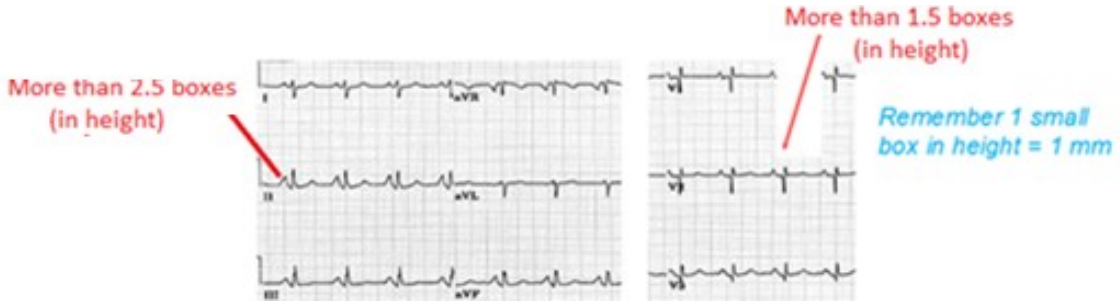
Lead II: P wave ›0.04 sec. (›1 small box) between notched peaks, or Lead V_1: -ve deflection ›1 box wide X 1 box deep.

Take a look at this ECG. What do you notice ?

The P waves in lead II are notched (**>**1 small box) & in lead V_1 they have deep & wide -ve deflection (**>**1 box wide X 1 box deep).

☆ **To diagnose *Right atrial enlargement,*** you can use the following criteria:-

Lead II: P **>** 2.5 mm, or Lead V_1 or V_2: P **>** 1.5 mm



The above ECG strip shows RAD (R wave -ve in lead I, +ve in lead II), peaked P wave P**>** 2.5 mm in lead II, P **>**1.5 mm In Lead V_1, V_2.

Management

(A) General measures

Dietary sodium restriction: should be implemented in all pts é CHF to **<** 3 gm/day. ***Activity & life style modification***: meals should be small in quantity but more frequent, reduce anxiety/emotional stress, avoid excess physical exertion (exercise may be advised within the limit of the pt's cardiac function), Wt loss is encouraged in obese pts, cessation of smoking & avoidance of other CVD risk factors.

(B) Control of the congestive state

Diuretics: useful in relieving congestion & reduce or prevent oedema. Most pts é HF have some degree of symptomatic congestion & benefit from diuretic therapy. Usually a loop diuretic is required é the addition of thiazide diuretic in pts refractory to the loop diuretic alone. *Furosemide*: initial dose 20-40 mg PO 1-2 tab daily or 20 mg IV, maximum dose 400 mg PO/day or 80 mg IV/day. *Hydrochlorothiazide*: initially 25 mg PO/ day, maximum dose 100 mg PO/day. The Loop & Thiazide diuretics are useful for symptomatic relief, however they have not been shown to improve survival. Their side effects include: azotemia, hypo-kalemia, metabolic alkalosis & elevation of neurohormones. *Spironolactone*: is an Aldost-erone inhibitor, reduces mortality in pts é advanced HF. This drug should be reserved for pts é moderate to severe HF (class IV symptoms), 25 mg PO/day or every other day, maximum dose 50 mg PO BID. Side effects; Hyperkalemia is common so monitoring K^+ level is essential. As a result, this drug should not be used in pts é a creatinine level >2.5 mg/dl. Gynecomastia in men is another side effect of this drug.

(C) Enhancement of myocardial contractility

Digoxin: is a drug ẃ has Inotropic effect; acts by inhibiting the Na^+- K^+ ATPase &⇧ intrace-llular Ca^{++}, this ⇧ myocardial contractility. In addition to *Neurohormonal modulation* of centrally mediated parasympathomimetic & sympatholytic activity, by doing so it blocks the AV node & delays AV conduction. Initially 0.125 mg PO/ day, maximum 0.25 mg PO/ day, tab 0.25 mg, syrup 0.05 mg%, amp 0.5 mg/2 ml. Start é digitalizing dose over 24 hrs, 20ug/Kg/day, 1^{st} dose is 50%, after 8 hrs 2^{nd} dose 25%, after 8 hrs 3^{rd} dose 25%, then maintenance dose equal to ¼ of the digitalizing dose (5ug/Kg÷2/day). It takes about 1 wk to digitalize pt. The IV digitalizing dose is 80% of oral dose. The tab. is well absorbed through GIT & initial effect can be seen within 30 min after oral administration, adjust the

dose in pt é renal failure, give ½ total digitalizing dose immediately & the succeeding 2 quarter doses at 12 hrs intervals & do ECG monitoring. The dose of digoxin almost never ⇧ but may ⇩ in presence of toxicity or RF. The signs of cardiac toxicity include; arrhythmia, bradycardia, AV block, PVCs, (premature QRS complex of abnormal shape & duration), also hypokalemia & hypercalcaemia ⇧ the toxicity of digoxin & in such condition discontinue the drug. The use of digoxin can improve symptoms, reduce the duration & the need for hospitalization in pt é HF, but has no effect on long term survival. Digoxin is renal excreted &so the dose adjustment is necessary in RF as mentioned above. A low dose of the drug (0.125 mg daily) should be prescribed, especially in women. The Digoxin use is recommended for pt é LV systolic dysfunction, particularly if they have AF & it is relatively contraindicated in some cardiac disease e.g. cardiac outflow obstruction in MS (in absence of AF) or corpulmonale. Because of its narrow window of safety, digoxin is associated é different side effects including:- anorexia, nausea, vomiting, wt loss, neuralgia, delirium, yellow vision, gynecomastia, arrhythmias of different types. If toxicity occurs, drug should be withheld & pt to be observed for some days before reinitiate é a lower dose. Electrolyte disturbances should be suspected & one should also give KCl, as hypokalemia ⇧ toxicity.

Vasodilators: may be useful in pts é severe acute HF who demonstrate systemic vasoconstriction despite ACEIs therapy. The vasodilators reduce the peripheral resistance & after load & improve cardiac performance. Hydralazine: initially 25 mg PO TID, maximum dose 150 mg PO QID. Isosorbide Dinitrate: initially 10 mg PO TID, maximum dose 80 mg PO TID. Hydralazine & nitrates in combination are effective afterload reducing agents used in ACEIs intolerant pts.

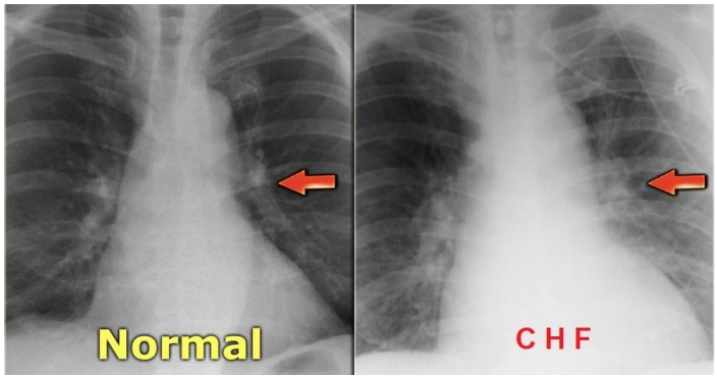
(D) Prevention of deterioration of myocardial function

The following drugs prevent deterioration in myocardial function by inhibiting the neuro-humeral mechanism ẃ causes cardiac remodelling & progression of HF.

1)Angiotensin converting enzyme inhibitors: cause reduction of the afterload & neurohormonal modulation as; *Captopril, Enalapril*, etc., have been shown to improve mortality, symptoms & hospitalizations. The dose of ACEIs should be titrated to the maximum that can tolerated symptomatically or the target dose. Initial dose of Captopril 6.25 mg PO/ day or every other day & its maximum dose 50-100 mg PO QID. Enalapril 2.5 mg PO BID & maximum dose 10-20 mg PO BID. The side effects of ACEIs include, angioedema, ARF in pt é bilateral renal artery stenosis. Other side effect is cough. The first two side effects are serious & necessitate immediate cessation of the drug. ACEIs are contraindicated é; angioedema, anuric RF, pregnancy & hypotension.

2) Angiotensin-II Receptor blocker: useful in pts who cannot tolerate ACEIs due to different side effects like cough, angioedema & leukopenia. *Losartan*: 25-50 mg 1-2 tab/day.

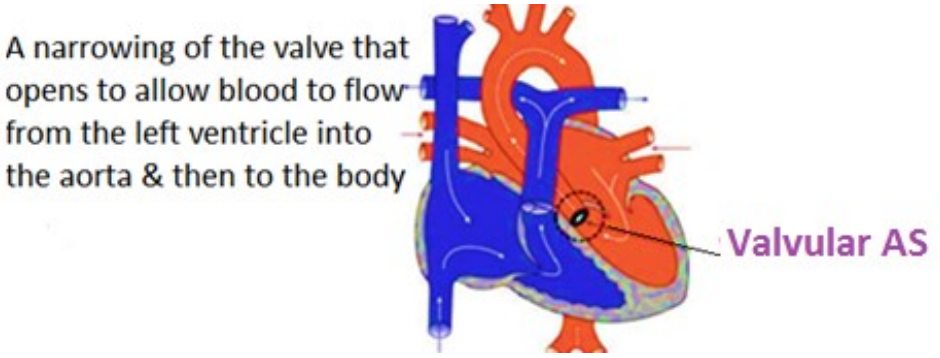
3) ß-adrenoreceptors blockers: administration of these drugs é gradual ⇧ of the dose has been reported to improve symptoms of HF, the need for hospitalization & reduce mortality. Indicated for moderate to severe HF. Contraindicated in unstable HF, hypotensive states, severe fluid overload, sinus bradycardia, AV block & asthma. *Metoprolol*: initial 6.25 mg PO BID & maximum dose 75 mg PO BID.



VALVULAR HEART DISEASES

VHD from chronic rheumatic fever (RF) is still the commonest cardiac disease in the developing world, occurring at the younger age. It causes significant morbidity & mortality due to lack of appropriate preventive & therapeutic intervention. Generally, pt é stenotic valvular lesion can be monitored clinically until symptoms appear. In contrast, pts é regurgete valvular lesions require careful ECHO monitoring for LV function & may require surgery even if no symptoms are present. Aside from antibiotic prophylaxis, very little medical Rx is available for pts é valvular heart disease. Surgery is the Rx for most symptomatic lesions or for lesions causing LV dysfunction even é absence of symptoms.

AORTIC STENOSIS



AS could be caused by: •Rheumatic cardities. •Congenital stenosis of aortic valve.•Senile /calcific AS ẃ is idiopathic results in calcification & degeneration of the aortic leaflets. •Persons born é bicuspid aortic valve are predisposed to develop AS. The aortic valve area must be reduced to one-fourth of its normal size before significant changes in the circulation takes place. Occasionally, the obstruction does not involve the aortic valve itself but consists of narrowing of the passage either above (supravalvular) or below it (subvalvular) caused by accumulation of fibroela- stic tissue, or calcification.

Clinical features

Initially there is an extended latent period during ẃ the pt is asymptomatic. This is

followed by the classic symptoms of AS including:-

- Angina.
- Exertional syncope.
- Dyspnoea & PND.

Physical examination

Systolic ejection murmur at 2nd LICS that radiates to the neck. In mild AS, the murmur peaks early in systole, but as the severity of AS ⇧, the murmur peaks progressively later in systole & may become softer as COP ⇩. As the stenosis worsens, the aortic component of the 2nd HS may become diminished. The timing & amplitude of the carotid pulse corre-late é the severity of AS. Later in the disease, the carotid upstrokes become diminished & delayed. ECHO: provides an accurate assessment of AV area & transvalvular gradient & also can be used to estimate LVH & EF. CXR: may demonstrate valve calcification.

Management

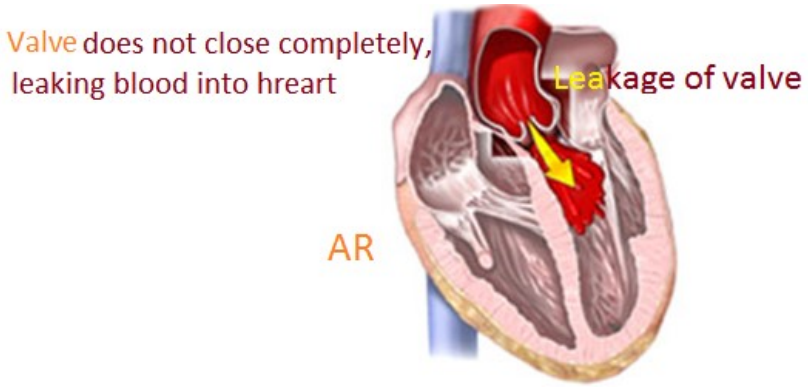
Medical Rx: is not effective & Rx é Digitalis or cautiously administered diuretics may only reduce symptoms. Pts é severe AS should limit vigorous physical activity. Pts é AS are at moderate risk for development of endocarditis & should receive endocarditis prophylaxis before selected procedures.

Surgical Rx: aortic valve replacement is the only effective Rx.

Prognosis

The survival of pt é AS is nearly normal until the onset of symptoms, when survival ra-tes ⇩ sharply. Although the rate of progression of AS is variable & difficult to predict, about 75% of pts é AS will be dead 3 yrs after the onset of symptoms if the aortic valve is not replaced.

AORTIC REGURGITATION



Defect in aortic root or aortic leaflets, preventing their normal closure. In chronic AR, the stroke volume⇧, ẃ in turn causes systolic hypertension, ⇧ pulse pressure & ⇧ afterload ẃ may be as high as that occurring in AS. The pt may be asymptomatic until severe LV dysfunction occur.

Causes

•Endocarditis. •Rheumatic fever. •Collagen vascular diseases. •Aortic dissection. •Syphilis. •Bicuspid aortic valves are also prone to AR.

Clinical features

The initial signs are subtle & may include ⇩ functional capacity or fatigue. As the disease progresses, the typical presentation is that of left-sided heart failure:-

•Orthopnoea. •Dyspnoea. •PND.

Physical examination

Diastolic blowing murmur heard along the LSB is characteristic of AR.

Diastolic rumble may also be heard over apex.

Peripheral signs of hyperdynamic circulation indicate severe disease, some of these signs include:- oWide pulse pressure oCollapsing pulse oQuincke's pulse (alternating blanching & erythema of the nail bed é gentle pressure applied). oDe musset's sign (head bobbing) oPistol shot over the femoral artery.

ECHO: provides information about aortic valve & aortic root size & semi quantitative estimate of the severity of AR & information about LV size & function.

Management

Systolic dysfunction is initially reversible é full function after AV replacement. Over time, however, progressive chamber enlargement é ⇩ contractility make recovery of LV function impossible, even é surgery.

Medical Rx

•**Diuretics** •**Salt restriction** •**Digoxin**: may be indicated in pt é severe regurgitation & dilated LV éout frank LV failure **Vasodilators**: afterload reduction é vasodilators has been shown to improve LV performance & reduce AR. The ACEIs are the preferred vasodilators. Rx é long acting **Nifedipine** in particular has been shown to delay the need for surgery by 2-3 yrs. Endocarditis prophylaxisis essential for all pts.

Surgical Rx

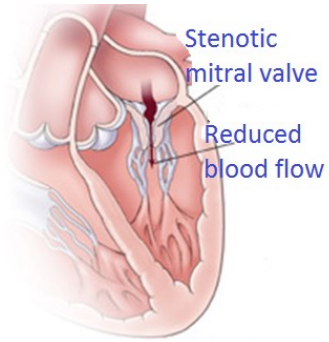
Aortic valve replacement is definitive Rx for pt é AR. Two important points to consider in deciding timing of surgery:-

a) Pt é AR usually don't become symptomatic until after the development of myocardial dysfunction.

b) When delayed too long, surgical Rx often does not restore normal LV function.

Therefor appropriate timing is necessary for surgical intervention. AR should be corrected if the symptoms are more than mild.

MITRAL STENOSIS



MS is squealy of RH primarily affecting women. MS has a progressive, life long course that is slow & stable in the early years é rapid acceleration later in life. It is very common in the developing countries manifesting below the age of 20 years. Elevated left atrial pressure eventually causes pulmonary vasoconstriction, pulmonary hypertension & compromise of right ventricular function.

Clinical features

Many pts remain asymptomatic until AF develops or until pregnancy occurs, when there is ⇧ demand on the heart. Symptoms are generally those of left sided HF.:-

- Orthopnea.
- Dyspnoea.
- Fatigability & PND.

Pt may also present é hemoptysis, signs of right-sided HF & embolic like stroke.

Physical examination

Apical rumbling, mid-diastolic murmur are characteristic & will immediately follow an opening snap, if present. The rumble is loudest in early diastole, but in pt é mild MS or MS é low COP, the murmur may be difficult to hear. It can accentuated by placing the pt in the left lateral decubitus position & using the bell of the stethoscope. Brief exercise (as walking in the hallway) may also accentuate the murmur. Loud 1^{st} HS is common. A right ventricular lift, elevated neck veins, ascites & oedema are later signs of right ventricular overload é pulmonary hypertension.

Complication

•AF. •Thromboembolism. •Right sided heart failure.

Investigations

ECHO: the study of choice for diagnosing &assessing severity of MS.

CXR: may show left atrial enlargement & sign of pulmonary congestion.

Management

Asymptomatic pt.

•Annual evaluation (history, physical exam, CXR & ECG).

•Endocarditis prophylaxis.

•Prophylaxis for rheumatic fever.

Symptomatic pt.

Diuretics are helpful in reducing left atrial pressure.

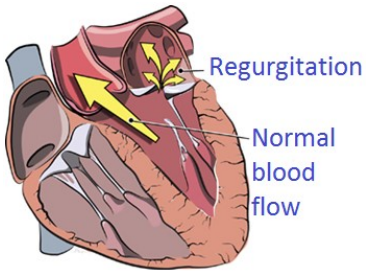
Digoxin indicated for pt é AF to control HR, since tachycardia will further ⇩ left ventricular filling, ⇩ COP & ⇧ left atrial pressure.

Anticoagulants warfarin indicated in pt é AF.

Surgical Rx

Improves survival & reduce symptoms, include, open commissurotomy, mitral valve reconstruction or replacement.

MITRAL REGURGITATION



Causes

•Rheumatic fever •Infective endocarditis •Degenerative valvular disease (mitral valve prolapse) •MI affecting papillary muscles.

Pathophysiology

Chronic MR is a state of volume overload leading to development of LVH. The Lf. atrium also enlarges to accommodate the regurgitate volume. This compensated phase of MR varies in duration but may last many yrs. The prolonged state of volume overload may eventually lead to decompensate MR. This phase is characterized by impaired LV function,⇩ EF + pulmonary congestion.

Clinical features

•Left sided HF: Fatigue, Excertional dyspnea, Orthopnea are the most common.

•Right sided HF é painful hepatic congestion.

•Peripheral oedema may occur in pt who have associated pulm hypertension.

Physical Examination

Soft 1st HS & wide split of the 2nd HS may present.

S3 gallop indicates severe disease but does not necessarily indicate HF. There may be displacement of the LV impulse.

Holo systolic murmur that may radiate to axilla & upper sternal border or the subscapular region.

Investigations

**ECHO:* used to determine the etiology & morphology of MR, ẃ are important in determining suitability for M. valve repair.

**CXR:* enlargement of LA & LV, pulmonary venous congestion, interstitial oedema & Kerley-B lines.

Management

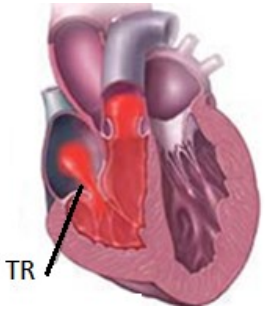
Medical treatment

- Diuretics.
- Salt restriction.
- Digoxin: may be indicated for pt é sever MR & dilated LV éout frank LVF.
- Vasodilators; afterload reduction é vasodilators has been shown to improve LV performance. ACEIs are the preferred vasodilators.
- Rx of AF if it occurs.
- Endocarditis prophylaxis is important essential.

Surgical Treatment

MV replacement is the definitive Rx. In pt é chronic MR, LV damage can occur while the pt remains asymptomatic. Therefore, surgery is indicated if LV dysfunction has begun to develop, even in the absence of symptoms. Pt é MR who is asymptomatic & whose LV function are normal are not considered for surgical Rx.

TRICUSPID REGURGITATION



TR is functional & secondary to marked dilatation of tricuspid annulus. It`s most common cause is pulmonary hypertension as a result of:-

☆ **LHF or pulmonary parenchymal/vascular disease**

☆**Less common causes include; Rheumatic Heart, Right side MI, or Endocarditis.**

Clinical features

Symptoms of right sided heart failure, peripheral oedema & ascites.

Pt will have prominent jaguar venous distension.

Presence of Holosystolic murmur at the LLSB.

Pulsatile liver & prominent ascites than oedema is common.

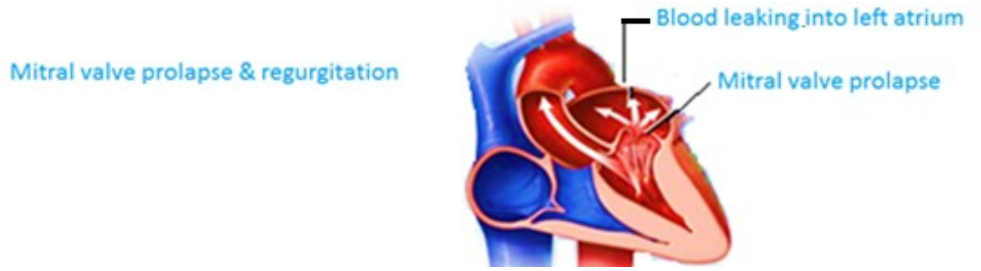
Diagnosis

•**Clinical examination** •**ECHO:** very useful study & differentiates 1ry from 2ry TR.

Management

- Rx of the underlying cause of HF usually reduces the severity of functional TR.
- Surgical treatment as indicated for primary TR.

MITRAL VALVE PROLAPSE



MVP occurs when varying portions of one or both leaflets of the mitral valve extend or protrude abnormally above the mitral annulus into the left atrium. MVP has different causes as redundant or excessive MV tissue, or congenital diseases as Marfan's sy or Osteogenesis Imperfecta. Although the prevalence of MVP was once thought to be as high as 15% of the general population, more recent studies using new ECHO criteria have suggested a prevalence of approximately 2.4%.

Clinical features

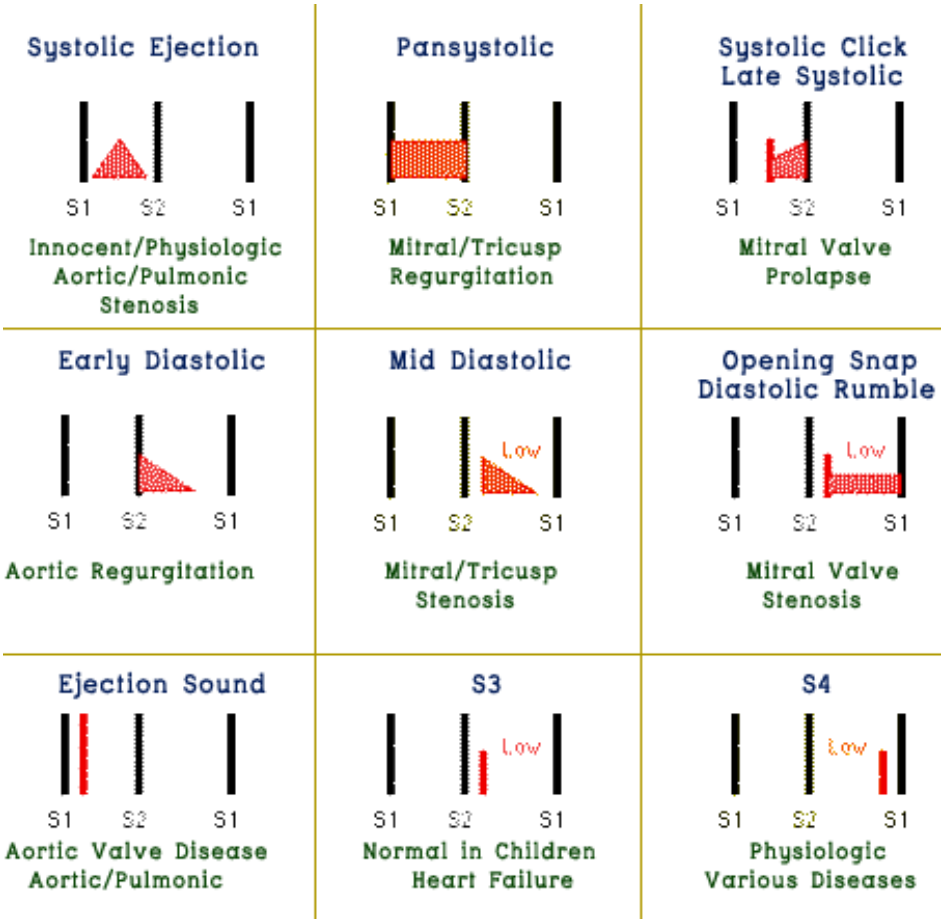
MVP is more common in females & in the age group of 14-30 yrs. The clinical course is often benign. Most pts are asymptomatic, may remain so for entire lives. Some pts may manifest é features of MR. Arrhythmias like PVCs & ventricular tachycardias may occur as complications. The mid-systolic click, often accompanied by late systolic murmur, is the auscultatory hallmark of MVP.

Management

Asymptomatic pts may need only reassurance. Symptomatic pt é thickening of MV will need endocarditis prophylaxis. ß-blockers sometimes may relive chest pain. Pt é severe symptoms from secondary MR, surgical Rx may be needed (mitral valve repair & or rarely replacement).

SUMMARY of VALVULAR HEART DISEASES

Even though the definitive Rx for most valvular heart diseases is surgical intervention to correct the underlying valvular abnormality. The onset of symptoms in the developing world occurs at an earlier age because of repeated attack of recurrent rheumatic fever. ⇧ Lf atrial pressure &⇩ COP produce the symptom. Therefore both 1ry & 2ry preventions are paramount importance to ⇩ the morbidity & mortality. In fact great progress has been made in improving rates of morbidity & mortality in pts é valvular Ht disease. Successful Rx of pts é valvular Ht disease requires an evidence-based approach to ECHO & to surgical intervention. ECHO should assess not only the valvular lesion but also the compensatory changes of the heart in response to the lesion. The timing of surgical treatment often correlates é outcome. Most pts é acquired valvular Ht disease are at risk for endocarditis.



INFECTIVE ENDOCARDITIS

Infection of the endocardial surface of the heart. The intracardiac effects of this infection include; severe valvular insufficiency, ẃ may lead to intractable CHF & myocardial abscesses. IE affects not only the heart, but also produces a wide variety of systemic signs & symptoms through several mechanisms, including both sterile & infected emboli & a variety of immunological phenomena. Common site of infection is heart valve, but may occur at septal defect or on chordae tendinae or in the mural endocardium.

Epidemiology

Incidence: **<**1% of the general population & it is more common in men, median age 50 yrs. Population groups at greater risk include the following:-

•RF history (most often involving aortic & mitral valves). •Hemodialysis. •Previous history of endocarditis or Pt é prosthetic valves. •IV drug users; 30% risk in in 2 yrs.

Etiology

Streptococcus viridans: is a bacterium ẃ is a normal flora of oral cavity. Accounts for 50-60% of cases of SIE. Group D streptococci; the source for this bacterium is the GIT or genitourinary tract. Most cases of IE due to this organism are SIE.

Staph Aureus is the leading cause of prosthetic valve IE & endocarditis in IV drug abusers. 35-60% of staph bacteremia is complicated by IE. In **>** half of cases of IE due to staph aureus occurs in the absence of underlying valvular disease, the mortality rate may range 40-50%.

Coagulase -ve Staph Aureus accounts for 30% of prosthetic valve IE & fewer than 5% of native valve endocarditis cases, causing SIE.

HACEK organisms accounts for 5% of IE. Include; Haemophilus, Actinobacillus, Cardiobacterium hominis, Eikenella corrodens, Kingella species. Usually cause SIE.

Fungus may cause IE, the most frequent cause is Candida Albicans.

Pathophysiology

All cases of IE develop from a commonly shared process include:-

(1) Bacteraemia (nosocomial or spontaneous) that delivers the organisms to the valves.

(2) Adherence of the organisms to valvular structures.

(3) Eventual invasion of valvular leaflets & formation of vegetations.

IE develops most commonly on the mitral valve, closely followed in descending order of frequency by the aortic valve, the combined mitral & aortic valves, tricuspid valve & rarely pulmonic valve. Mechanical prosthetic & bioprosthetic valves exhibit equal rates of infection. Turbulence in blood flow damages valvular surface & endocardium ẃ creates favourable situation for formation of sterile thrombus (vegetation) made of platelets & fibrin. The microorganisms that most commonly produce IE (strept viridans, staph aureus, group A, C, D strept & enterococci) resist the bactericidal action of complement. The result of an invasive procedure gives access to the bacteria to adhere to the sterile platelet /fibrin vegetation. Most cases of SIE are secondary to the bacteremia that develop from the activities of daily living (brushing teeth, bowel movements). The complications of acute te IE result from intracardiac disease & metastatic infection produced by suppurative emboli.

When to suspect?

(a) Sepsis of unknown origin.

(b) Fever coexisting é:- •Intracardiac implantable material. •Congenital heart or valve disease. •Presence of IE risk factors •Presence of CHF symptoms. •New heart block. •Positive blood cultures. •Peripheral abscesses (of kidney, spleen…).

Classification

Native Valve Endocarditis (NVE): develop on natural valve. The affected valve may be damaged or normal.

Prosthetic Valve Endocarditis: develops on prosthetic 'artificial' valve.

Endocarditis in IV Drug Abuser: marked ⇧ in cases of prosthetic valve endocarditis

Clinical course

Subacute Infective Endocarditis

Typically affects only previously damaged valves, has insidious course ẃ may extend over many months. The pt suspected to have SIE should be asked about invasive procedures that may cause bacteremia. Mostly caused by St. Viridans & related to dental disease. Symptoms of early subacute NVE usually are subtle/nonspecific, suggested by a history of a slowly progressive process characterized by fever, fatigue, anorexia, back pain & wt loss. Less commonly are stroke or CHF. When appropriate Rx is delayed for wks or months, additional clinical features, either embolic or immunological in origin, develop.

The embolic manifestations include:-

•Acute meningitis é sterile spinal fluid.

•Hemiplegia due to embolization in the distribution of the middle cerebral artery. •Renal regional infarcts producing painless hematuria.

•Splenic infarction. •Unilateral blindness caused by occlusion of a retinal artery.

•MI from embolization to coronary artery. The risk is related to type of organism, size of the vegetation & rate of growth or resolution & location of vegetation.

The immunologic manifestations include:-

•Acute glomerulonephritis. •Osler's nodes.

•Roth spots. •Presence of Rh^{ed} factor.

Physical findings

Fever: most pts have low grade fever; however 3-15% of pts may have normal or subnormal temperature.

Murmurs**:** the vast majority of pts have detectable murmurs (99% of cases). The absence of murmur should cause clinician to reconsider the diagnosis of IE. The major exception is right sided IE in ẃ only 1/3 of cases have a detectable murmur. ***The peripheral lesions:*** observed in only 20% of pts as compared to 85% in the preantibiotic era, including:-

Petechiae ẃ is the most common (result of fragmentation & microembolization of vegetative lesions), may occur on the palpebral conjunctivae or dorsum of hands & feet, or anterior chest & abdominal wall or oral mucosa & soft palate.

Subungual Hge: black longitudinal streaks not extending to the entire length of nails.

Clubbing of fingers & toes: may be seen primarily occurs in pts who have an extended course of untreated IE.

Arthritis: is asymmetrical, limited to 1-3 joints.

Splenomegaly: observed more commonly in pts é long standing SIE & may persist long after successful therapy.

Osler nodes: are small tender nodules pea-size lesions that range in color from red to purple, located primarily in the pulp spaces of terminal phalanges of fingers, toes, soles of feet, thenar & hypothenar eminences of the hands.

Acute Infective Endocarditis

Frequently involves healthy valves, is a rapidly progressive é destruction of valvular structures. History of antecedent procedures/illicit drug use must investigated. Is a much more aggressive disease. The onset of illness is abrupt é rapidly progressive destruction of the infected valve. The valvular leaflets are destroyed rapidly by bacteria that multiply

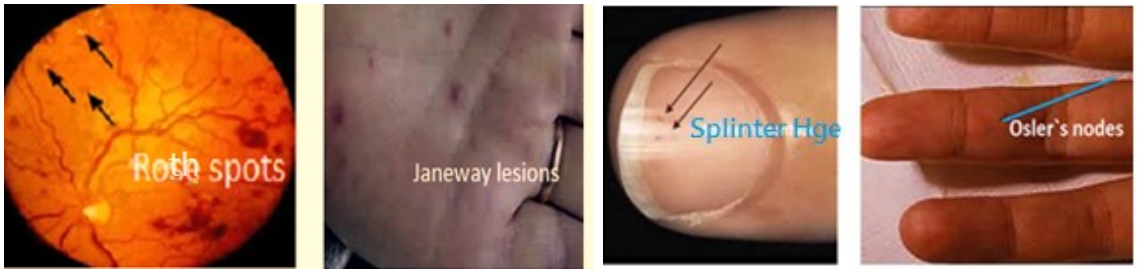
very fast within the ever growing friable vegetations.

The clinical symptoms of AIE result from either embolic or intracardiac suppurative complications. Characterized by acute onset of ***high-grade fever, chills*** & rapid onset of ***CHF.***

Complications of AIE

Develop within a week, include:-

- ***Dyspnoea & Fatigue*** of severe CHF.
- ***Neuropsychiatric complications:*** resulting from involvement of the CNS.
- ***Roth spots:*** retinal Hge é pale centers. Litten sign "cotton-wool exudates".
- ***Murmurs:*** present in 2/3 of pts. The most common is murmur of AR. Because of the rapid onset, the LV does not have chance to dilate. In this situation, the classic findings of ⇧ pulse pressure that are seen in chronic AR are absent.
- ***Janeway lesions:*** irregular erythematous & painless macules (1-4mm in diameter). Most often located on the thenar & hypothenar eminences of hands & feet. They represent an infectious vasculitis of AIE resulting from staph aureus infection.
- ***Acute septic monoarticular arthritis:*** most often caused by staph aureus infection.
- ***Purulent meningitis:*** observed in pt é AIE, as compared to the aseptic type observed in pt é SIE.



The right-sided infective endocarditis

Is associated é a very low rate of CHF & valvular perforation. Pulmonary infarcts may result from emboli of right-sided IE.

The percentage of occurrence of clinical & laboratory manifestations

Clinical manifestations	*% of occurrence*
Fever	> 95
Arthralgias &/or myalgias	25-45
Murmur	> 85
Splenomegaly	25-60
Splinter haemorrhages	20-40
Roth's spots	< 5
Osler's nodes	10-25
Janeway lesions	< 5
Clubbing	10-20
Clinically apparent emboli	25-45
Neurologic manifestations	20-40
laboratory manifestations	%
Anaemia	70-90
Leucocytosis	20-30
Proteinuria	50-65
Microscopic haematuria	30-50
Elevated s. creatinine level	10-20
Elevated ESR.	>90
Rheumatoid factor	50
Circulating immune complexes	65-100
⇩ serum complement level	5-40

Diagnostic work up

• ***Blood Culture:*** gold standard test for diagnosis of IE is documentation of a continuous bacteremia (**>**30 min in duration) through blood culture. For making diagnosis of SIE, draw 3-5 sets of blood cultures, at 3 different sites, over 24 hrs. This detects 92-98% of cases in pts whom did not receive antibiotics recently. In the case of AIE, 3 sets may be drawn over 30 min (é separate vein punctures) to document a continuous bacteremia.

• ***ECHO:*** has become the indirect diagnostic method of choice, especially in pt who presents é clinical picture of IE but who have non diagnostic blood cultures. TTE & TEE to see vegetation's on mitral or aortic valve, or other findings as abscess, pseudoaneurysm, per-

foration, fistula, valve aneurysm, or dishence of prosthetic valve. TEE is 1st choice to find IE complications. Sensitivity of TEE is bigger than TTE (90-100% vs 40-63%). But diagnosis of IE can never excluded by -ve ECHO.

- ***ECG:*** detect 10% of pts who develop conduction delay (prolonged P-R interval).
- ***Rhed factor:*** becomes +ve in 50% of pts é SIE, it becomes -ve after successful Rx.
- ***WBCs*** é differential.
- ***ESR & CRP.***

Duke criteria

Major Criteria
•Positive Blood culture: for typical microorganism that causes IE from 2 separate blood cultures (strept. viridans, strept bovis, HACEK group, or community-acquired staph aureus or enterococci in the absence of a primary focus). •Positive ECHO: include:-Definitive vegetation (oscillating intracardiac mass on valve) or Abscess, or New partial dehiscence of prosthetic valve, or New valvular regurgitation (not changes in pre-existing murmur).
Minor Criteria
•Predisposing heart condition or IV drug abuse. •Fever >38.0 °C. •Embolic phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, ICHge, conjunctival Hge, Janeway lesions. •Immunologic phenomena: Glomerulonephritis, Osler's nodes, Roth's spots, or Rhed factor. •Microbiologic: positive blood culture but not meeting major criterion. •ECHO: consistent é IEbut not meeting major criterion.

**Definitive diagnosis made by documentation of: 2 major or 1 major + 3 minor or 5 minor.*

Management

General measures

Diet: no special diets recommended for pt é IE, however if the pt has CHF, sodium restriction may be necessary.

Activity: activity limitations are determined by the severity of IE, complications (e.g., stroke) & by the presence of significant CHF.

Medical treatment

The major goals of therapy for IE are:-

- Eradicating the infectious agent from the thrombus: antibiotics remain the mainstay of Rx for IE. In the setting of AIE, antibiotic Rx should be instituted as soon as possible to minimize valvular damage. 3-5 sets of blood cultures should be obtained within 60-90 minutes, followed by the infusion of the appropriate antibiotic regimen. By necessity, the initial antibiotic choice is empiric, determined by clinical history & physical examination. In the case of SIE, Rx may be safely delayed until cultures & sensitivities are available. Waiting does not ⇧ the risk of complications in this form of the disease. Eradicating bacteria from the fibrin-platelet thrombus is extremely difficult. IV administration is the preferred to ensure reliable serum therapeutic levels. The antibiotics should be bactericidal & should administered at higher dose for prolonged period of time. The *Empirical antibiotic Rx of SIE are:* Crystalline Penicillin 3-4 million U IV/4 hrs for 4-6 wks + Gentamicin 1 mg/ kg IV TID for 2 weeks. For *Prosthetic, valve endocarditis:* Vancomycin 1 gm IV BID for 6 wks + Gentamicin 1 mg/kg IV TID for 2 wks + Rifampicin 300 mg PO/ TID for 6 wks. For *AIE: where staph aureus suspected:* Naficillin 1.5-2 gm IV/4 hrs or Vancomycin 1 gm IV BID for 6 wks + Gentamicin 1 mg/kg IV TID for 2 wks. When the result of blood culture is made available the choice of antibiotics depend on the type of the organism & sensitivity.

•Dealing é the complications of valvular infection: includes both the intracardiac & extra-cardiac consequences of IE. Mild CHF resulting from valvular insufficiency or myocarditis may be managed é the standard medical Rx for CHF. Although thrombosis is a key element of IE, anticoagulation é Heparin or Warfarin is controversial & it should be avoided.

Surgical treatment

15-25% of pts é IE require surgery. The indication for surgery include:-

•AR é severe HF.

•Fungal endocarditis.

•Mobile vegetation >10 mm in size.

•Evidence of valve ring or myocardial abscess.

•Recurrent embolization despite adequate antibiotic Rx.

•Poor response to antibiotics.

•Prosthetic valve dysfunction é CHF or valve ring abscess near a prosthetic valve.

Admission to hospital

Pt should be treated in the hospital to allow adequate monitoring of the development of complications & the response to the antibiotic.

Complications

•CHF: ~60% of IE pts.

•Systemic embolism: 30% of pts (brain, spleen, lungs).

•Uncontrolled infection. •Neurologic events.

•ARF. •Rheumatic problems. •Myocarditis.

Prophylaxis

* First & most important- Proper oral hygiene & Regular dental review.

* Antibiotics reserved for narrow group of high risk pt.

MYOCARDITIS

Inflammation of the myocardium often resulting from infectious process, ẃ subsequently leads to myocardial destruction & dilated cardiomyopathy. The acute picture is nonspecific unless overt CHF develops. Although the causes are numerous, the most common association is an antecedent viral syndrome.

Etiology

Infectious causes

- **Viral**: Coxackie virus B, HIV (overt involvement seen in 10% of HIV pts).
- **Bacterial**: not common, usually occurs as a complication of IE. Diphtheric myocarditis may occur in 25% of pts é diphtheria.
- **Fungal.**
- **Protozoal**: Chagas disease caused by a trypanosoma cruzi & transmitted by an insect & is one of the common causes of heart disease in central & south America.
- **Rickettsial**: associated é Typhus, Lyme disease.
- **Spirocheatal**: associated é relapsing fever.
- **Hypersensitivity & toxic reaction*:*** Doxorubicin (antineoplastic) or radiation.

Giant cell myocarditis: is a rare form & of unknown aetiology.

Pathophysiology

Myocarditis defined as inflammatory changes in heart muscle & characterized by an interstitial mononuclear cell infiltrate é an attendant myocyte necrosis. It is not known whether the infiltrate is caused by direct invasion of the infective agents or by a systemic immune response. In the chronic stage, cytotoxic T lymphocytes infiltrate the myocardium & mediate an autoimmune response é myocardial autoantibody activity directed against cardiac myosin. This process persists after the viral particles are no longer detected.

Coronary artery thrombosis, luminal obstruction, ischemia & dysrhythmias compound the deleterious effects of inflammation.

Clinical features

The clinical presentation is variable. Pt may present é nonspecific illness characterized by fatigue, mild dyspnea, or fulminant CHF. Myocarditis may even cause sudden death in some pts. The majority of cases of myocarditis are subclinical; therefore, the pt rarely seeks medical attention during acute illness. An antecedent viral syndrome documented in 60% of cases. The typical time interval between the onset of viral illness & cardiac involvement is 2 wks. Fever is present in 20% of cases. Fatigue, myalgia & malaise are common. Chest pain or discomfort is reported in 35% of cases, it is often pleuritic quality é precordial pain of a sharp stabbing nature. Sometimes it may be substernal & squeezing, more typical of ischemic pain. Dyspnoea on exertion is common & orthopnea & shortness of breath at rest may be noted if CHF is present. Palpitations are common. Syncope signals development of AV block or malignant dysrhythmias & may lead to sudden death.

Physical examination

Pt é mild myocarditis has a nontoxic appearance & simply may appear to have a viral syndrome. Tachypnea & tachycardia are common. Tachycardiais often out of proportion to fever. More acutely ill pt have signs of LVF including: ⇧ JVP, bilateral basal crepitations, ascites & peripheral oedema. S3 gallop may be noted é significant cardiac enlargement (displaced apical impulse). S1 is soft or muffled & cyanosis may be noticed. Hypotension caused by LV dysfunction is uncommon in the acute setting. A poor prognosis is indicated when hypotension is present. Cardiogenic shock observed in fulminant cases é high mortality. Murmurs of mitral or tricuspid regurgitation may be present due to ventricular

dilatation .In cases where a dilated cardiomyopathy has developed, signs of peripheral or pulmonary thromboembolism may be found. Associated pericarditis may manifest é a pericardial friction rub. Pericardial effusion is common, but signs of tamponade (hypotension, jugular venous distension, muffled Heart sounds) are rare. Pleural friction rub might be heard as pleuritis can occur é acute myocarditis.

Diagnostic workup

Since many cases of myocarditis are not clinically obvious, a high degree of suspicion required for making diagnosis.

- ***ESR*** is ⇧ in 60% of pts é acute myocarditis.
- ***Leucocytosis*** is present in 25% of cases.
- ***CXR:*** often reveals normal cardiac shadow, but pericarditis or overt clinical CHF may be associated é cardiomegaly. Vascular redistribution, interstitial & alveolar oedema & pleural effusion may be seen on CXR.
- ***ECHO:*** dilated chambers & ⇩ EF indicating LV systolic dysfunction.
- ***ECG:*** sinus tachycardia is the most frequent finding. ST-segment elevation éout reciprocal depression, particularly when diffuse, is helpful in differentiating myocarditis from acute MI.

Treatment

Pt é mild symptoms & no signs of cardiac failure or dysrhythmia may be treated on an outpatient basis. The medical Rx is directed towards amelioration of associated complications including CHF, cardiogenic shock, arrhythmias & thromboembolism.

<u>Lf vent. dysfunction é signs of CHF should be treated é;</u>

•Low sodium diet. •Limitation or avoidance of exercise. •Diuretics. •Digoxin: sho-uld be

given é caution as pt é myocarditis is sensitive for digitalis toxicity. •ACEIs & vasodilators. In general, sympathomimetic & B-blocker drugs should be avoided because they ⇧ the extent of myocardial necrosis & mortality.

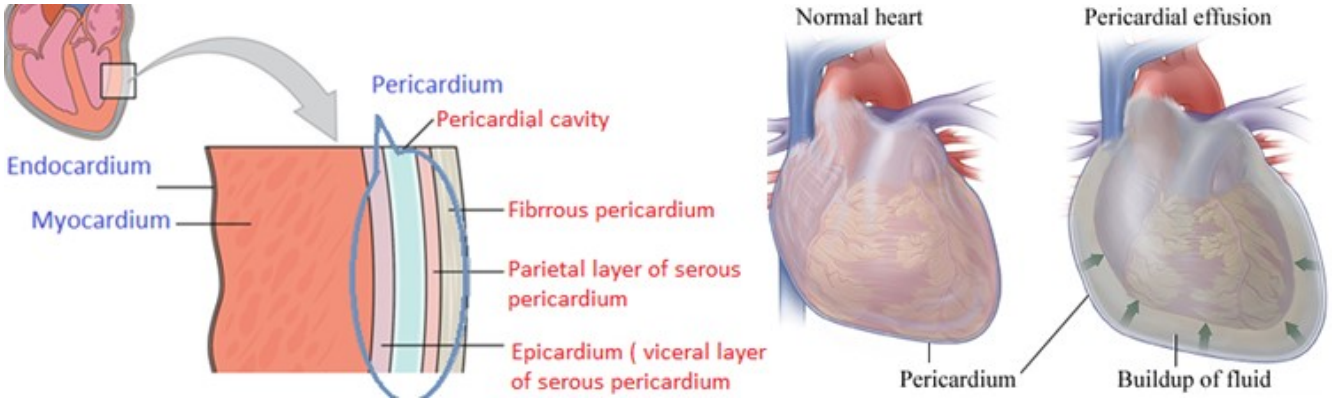
Arrhythmias: detection of dysrhythmia é inpatient cardiac monitoring. Medical Rx for arrhythmias or implantation of pacemakers.

Avoiding risk of thromboembolism: anticoagulants such as warfarin or heparin may be given. NSAID are contraindicated in the early course of the disease because of inhibition of prostaglandin production, worsened myocyte function & ⇧ myocardial necrosis.

Prognosis

Majority of cases are believed to be clinically silent & resolve spontaneously éout sequealy; therefore, it is difficult to make accurate statements concerning the prognosis of myocarditis. Pt presenting é CHF experience morbidity & mortality based on the degree of LV dysfunction & the presence of arrhythmias & thromboemblic complications ẃ ⇧ the risk of mortality.

PERICARDITIS & PERICARDIAL EFFUSION



Inflammation of the pericardium surrounding the heart. Pericarditis & cardiac tamponade are clinical problems involving the potential space surrounding the heart or pericardium. Pericarditis is one cause of fluid accumulation in this potential space & cardiac tamponade is hemodynamic result of fluid accumulation.

Pathophysiology

The pericardium consists of an outer fibrous layer (parietal pericardium) & an inner serous layer (visceral pericardium). Normally the 2 layers are separated by a small quantity of fluid (15-50 ml). The pericardium serves as a protective barrier from the spread of infection or inflammation from adjacent structures. It also prevents sudden dilatation of the cardiac chambers during exercise & hypervolemia. It restricts the anatomic position of the heart & minimizes friction é the surrounding structures. Approximately 120 cc of additional fluid can accumulate in the pericardium éout ⇧ in pressure. Further fluid accumulation can result in marked ⇧ in pericardial pressure, eliciting ⇩ in COP &⇩ BP (cardiac tamponade). The rapidity of fluid accumulation influences the hemodynamic effect.

Clinical picture

The most common symptom of acute pericarditis is precordial or retrosternal **chest pain**, usually described as sharp or stabbing. Pain may be of sudden or gradual onset & may ra-

diate to the back (left trapezial ridge), neck, left shoulder, or arm. Movement or inspiration may aggravate the pain. Pain may be most severe when pt is supine & can be relieved when pt leans forward while sitting. **Kussmaul sign**-neck veins distend é inspiration (constrictive type). **Low-grade intermittent fever, cough, dyspnea & dysphagia.** In TB pericarditis, fever, night sweats & wt loss seen in 80% of cases. ***Cardiac tamponade:*** as the volume of pericardial fluid ⇧, the capacity of atria & ventricles to fill is mechanically compromised, leading to ⇩ stroke volume & tamp- onade. It is influenced by volume & rate of accumulation. ***Beck triad:*** jugular venous distension + hypotension + muffled HS. ***Neck vein distension:*** is common finding: but Kussmaul`s sign is -ve. ***Hypotension:*** to the extent of shock: may occur when cardiac stroke volume significantly ⇩ & tissue perfusion is compromised. ***Pulsus paradoxus:***⇩ in BP ﹥10 mmHg during inspiration. ***Narrow pulse pressure*** indicates ⇩ LV stroke volume. ***Cyanosis. Altered consciousness:*** pt may present subacutely é symptoms of anxiety, dyspnea, orthopnea, fatigue. Pt may have a history of medical illnesses associated é pericardial involvement, particularly endstage renal disease. A waxing waning clinical picture may be present in intermittently decompressing tamponade. ***Ewart sign:*** dullness & bronchial breathing between the tip of the left scapula & the vertebral column. ***Hepatomegaly & ascites*** may be found.

Classification of pericarditis

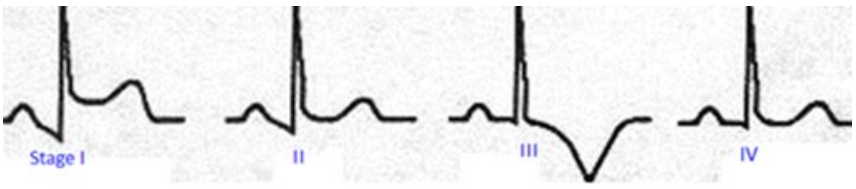
Clinical classification	Etiologic Classification
I. Acute pericarditis (< 6 weeks) Fibrinous - Effusive (serous or sanguineous) II. Subacute pericarditis (6 weeks- 6 months) Effusiveconstrictive - Constrictive III. Chronic pericarditis (> 6 months) Constrictive - Effusive - Adhesive	I. Infectious pericarditis Viral - Pyogenic - Tuberculous - Fungal II. Noninfectious pericarditis Uremia - Acute MI - Neoplasm - Idiopathic. III.Hypersensitivity/autoimmune Rh fever - Rh^{ed} arth - SLE - Posttraumatic.

TB pericarditis is quite common in HIV +ve pts. Viral & autoimmune causes represent ﹥50% of cases.

Diagnosis

2 of the following features including the ECG changes are necessary for diagnosis:-

- ***Chest Pain:*** described in detail é the clinical picture above.
- ***Pericardial friction rub:*** highly specific é variable sensitivity. A high-pitched scratchy or squeaky sound best heard é the diaphragm at the LSB é the pt leaning forward. Have 3 components, ẃ correspond to atrial systole, ventricular systole & early diastole. Audible throughout the respiratory cycle, whereas the pleural rub disappears when respirations are on hold. Friction rub may be transient from one hour to the next & is present in approximately 50-85% of cases. Friction rub may distinguished from a cardiac murmur by its changing character from heart beat to another & pt position changes, in addition that it is closer to the ear on auscultation than a murmur.
- ***ECG changes:*** Acute pericarditis present è widespread upward concave S-T segment elevation & P-R segment depression. If the ratio of S-T segment elevation to T wave amplitude in V6 **>** 0.24, acute pericarditis is almost always present. The ECG changes have 4 phases during the course of illness; each phase lasts from few days to few weeks, while phase IV lasts for several months for gradual resolution of T wave changes. Typical diffuse ST elevation not seen é uremic pericarditis, in ẃ fibrin is deposited in parietal layer é no epicardial inflammation.



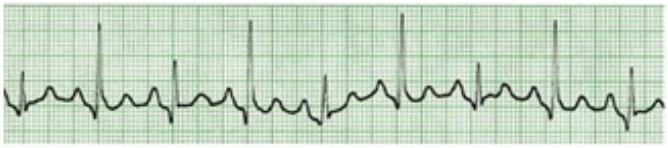
Stage I: everything is UP (i.e., ST elevation in almost all leads)

Stage II: transition (i.e., "pseudonormalization").

Stage III: everything is DOWN (inverted T waves).

Stage IV: normalization.

Cardiac tamponade or massive effusion: electrical alternans is pathognomonic & is characterized by alternating levels of ECG voltage of P wave, QRS complex & T waves. This is a result of heart swinging in a large effusion.

Electrical pulsus alternans 

- **CXR:** recommended in all cases. It is typically normal, or associated é enlarged cardiac silhouette in effusion (é clear lung fields). Bottle shaped heart may be seen é excessive pericardial fluid accumulation. In cardiac tamponade (or large effusions), CXR may demonstrate enlarged cardiac shadow after 200-250 ml of fluid accumulation. The chronicity of effusion may suggested by the huge cardiac shadow.
- ***ECHO:*** minimal pericardial effusion in pericarditis. Significant pericardial effusion in chronic pericarditis or cardiac tamponade. Presence of effusion supports the diagnosis, but absence does not exclude it.
- **Laboratory abnormalities:** ⇧ **WBC** (purulent pericarditis). ⇧ **ESR & CRP**. ⇧ **Uric acid** (uremic etiology). **HIV** in selected cases. **ANA & Rh^{ed} Factor. Blood cultures** if febrile. Viral cultures & antibody testing not indicated. **Cardiac isoenzymes**? helpful, **MB fraction of CK** & **Troponin 1:** are modestly ⇧ it is related to the extent of myocardial inflammation. Features associated é rise in (Tn-I) are younger age, male gender, presence of effusion & a recent infection. The enzyme rise is transient, resolving within the 1st wk, it`s persistent rise suggest myopericarditis. **TB skin test**

Differential Diagnoses

- MI.
- Myocarditis.
- Pulmonary embolism.
- Pneumothorax.
- Pneumopericardium.
- Musculoskeletal cause.

Need for hospitalization

Many physicians tend to admit them, but this may not be necessary. Uncomplicated acute pericarditis can undergo initial evaluation in a same day hospital facility or clinic é an outpatient follow-up.

Features of high risk

•Subacute symptoms (e.g. developing over several days or wks). •Fever (>38^{0}C) & leucocytosis. •Evidence of cardiac tamponade. •Large pericardial effusion •Immunosuppressed state. •History of oral anticoagulant therapy. •Acute trauma •Failure to respond within 7 days to NSAID. •⇧ Cardiac treponin-1.

Complications

•Recurrence in 15-32% of pts, mostly in cases of autoimmune etiology. •Pericardial effusion/cardiac tamponade. •Chronic constrictive pericarditis: can be "transient"- 10% may have transient within the 1st month & resolves by 3 months. •Cardiac perforation at time of pericardiocentesis. •Bronchopericardial fistula: noted as complication of multi drug-resistant TB inHIV pt.

Management

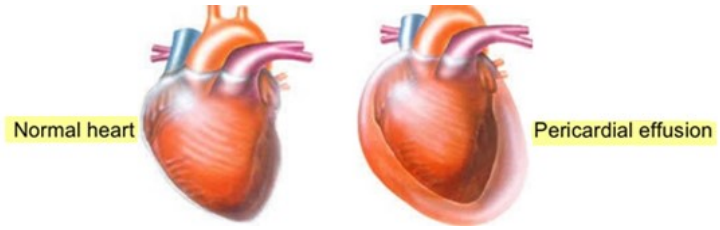
Goals of acute therapy: •Relieve pain. •Treat inflammation. •Prevent cardiac tamponade. •Treat underlying disease •Drain purulent effusions. •Symptomatic treatment.

Treating pain & inflammation: **NSAIDs** are effective in reducing the inflammation & reliving chest pain. May require wks to months of Rx é high doses. The choice of NSAND is usually empiric, based on the physician's familiarity é the agent &/or its availability. Rapidly titrate the dose within 1-2 days to achieve maximum symptomatic relief. Evaluate for a response within 1-2 wks, symptoms usually subside in a week. If adequate clinical response, continue NSAIDs for 1 wk after complete resolution of symptoms, then taper in

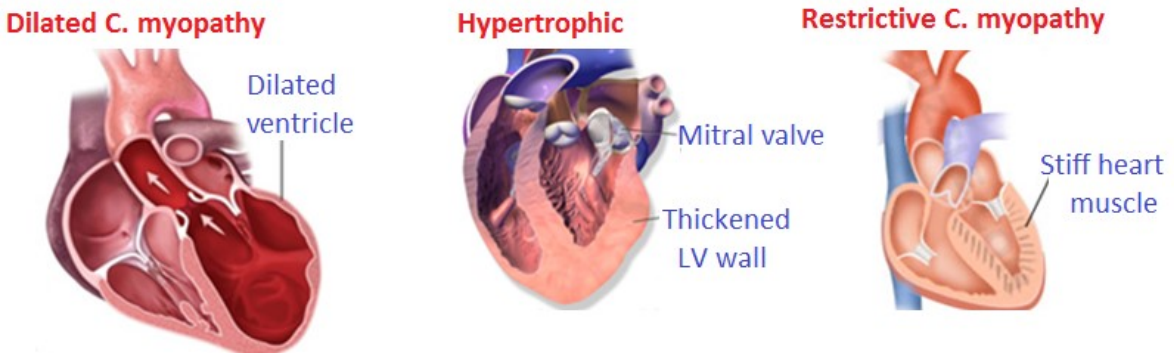
2-3 days.

Aspirin: 2-6 gm daily, preferred in pt é coronary artery disease. **Ibuprofen**: 400-800 mg q 6-8 hrs. **Indomethacin:** 75-225 mg daily, try to avoid, unless absolutely needed, it can ⇩ coronary blood flow. **Colchicine:** prospective, randomized, open-label design was used. 120 pts é a first episode of acute pericarditis were randomly assigned to conventional Rx é Aspirin (group I) or conventional Rx plus colchicine 1.0-2.0 mg for the first day & then 0.5-1.0 mg/day for 3 months (group II). Colchicine significantly ⇩ the recurrence rate (10.7 % vs 32.3 %; P= 0.004) & presence of symptoms at 72 hrs (11.7 % vs 36.7%;P= 0.003). Based upon this, addition of it to the Rx regimen for an initial episode of acute pericarditis is an option. **Steroids:** in pt refractory to NSAID & Colchicine. Steroid Rx é initial episode is more likely associated é recurrent episodes. Evidence available argues against the routine administr-ation of steroids during the 1st episode of acute pericarditis. Specific conditions that will benefit from steroids include; acute pericarditis due to connective tissue diseases, or autoimmune pericarditis & Uremic pericarditis.

Specific therapy: • ***TB pericarditis*** should be treated é anti TB along é steroid. • ***Cardiac tampon*ade:** pt will need emergency pericardiocentesis. • ***MI associated pericarditis:*** early post MI pericarditis is a consequence of transmural MI. Aspirin is drug of choice in this setting. Late MI associated pericarditis (Dressler sy.) occurs days- months after MI, is autoimmune in etiology & NSAIDs are the Rx of choice. Colchicine seems to be most eff-ective if NSAIDs fail. Corticosteroids seem to provide symptomatic benefit (not prevent recurrence). Pericardiectomy is only rarely curative.



CARDIOMYOPATHY



Group of diseases that affect the myocardium & are not the result of; hypertension, valvular, coronary or pericardial abnormalities. frequently associated é myocardial dysfunction & subsequently HF. Histologic findings are nonspecific é myocyte hypertrophy, cellular necrosis & fibrosis.

Etiologic classification

Primary myocardial involvement: •Idiopathic. •Familial. •Eosinophilic endomyocardial diseases. •Endomyocardial fibrosis.

Secondary myocardial involvement: •Infective (viral, bact., fungal, protozoal, spirochatal). •Metabolic (thyrotoxicosis). •Peripartum heart diseases. •Familial storage diseases (glycogen). •Deficiencies (Electrolyte, Vit B_1). •Connective tissue diseases (SLE, Rheumatoid). •Infiltration & granulomas (sarcoidosis, malignancies). •Neuromuscular (muscular dystrophies, myotonic dystrophies). •Sensitivity & toxicity.

Classification

Type	Description
Dilated	Dilatation & impaired contraction of the Lf or both ventricles. Caused by familial/genetic, viral, immune, alcoholic, toxic or unknown factors, or associated é cardiovascular disease
Hypertrophic	LVH &/or RVH, often asymmetrical, usually involves the interventricular septum. Mutations in sarcoplasmic proteins cause the disea-se in many pts.
Restrictive	Restricted filling & reduced diastolic size of either or both ventricles é normal or near-normal systolic function. Is idiopathic or associated é other diseases.

DILATED CARDIOMYOPATHY

Impaired left &/or right ventricular systolic function. characterized by an EF ˂40% in the presence of ⇧ LV dimensions (LV end-diastolic size ˃115% of that calculated for age & body surface area).

Pathophysiology

Dilated cardiomyopathy represents the final common morphologic outcome of a variety of biological insults. It is a combination of myocyte apoptosis & necrosis é ⇧ myocardial fibrosis, producing ⇩ in mechanical function. Many causes are a result of direct toxicity (e.g. alcohol) or mechanical insults (e.g. chronic volume overload in MR), or infection (e.g. in myocarditis).

Clinical manifestations

Careful history is essential é particular emphasis on; family history of similar illness. Exposure to cardiotoxins such as alcohol. Protracted "flulike illness" or RTI may suggest previous myocarditis. History of recent delivery or being in last TM of pregnancy. Some pts may have LV dilatation for months or even yrs & may remain asymptomatic & diagnosed only by screening or post-mortem examination. Symptoms of **Lf & Rt sided CHF** develop gradually in most cases. Unfortunately, the commonest clinical presentation is one of progressive deterioration é worsening HF & death occurring over a variable time course. **Syncope** may result from **arrhythmias**. **Systemic embolization**, often emanating from ventricular thrombus..

Physical examination

•Hypotension. •Tachycardia. •Cardiomegaly. •Findings of CHF: narrow pulse pressure , ⇧ JVP & S3, S4 gallops. •Functional mitral or tricuspid regurge.

Diagnostic work up

- **CXR:** cardiac enlargement, evidence of pulmonary congestion.
- **ECG:** sinus tachycardia or AF, ventricular arrhythmias, ST segment abnormalities.
- **ECHO:** LV dilatation & dysfunction (EF 〈40%).
- **Other investigations :-**CBC, RFTs, FBS, PPBS, lipid profile & TFTs.

Treatment

Medical Rx: standard Rx of HF include:-

•Salt restriction. •Diuretics (spironolactone). •Digitalis. •ACEIs or ARBs. •Alcohol sho-uld be avoided. •Identify &treat the underlying cause if it is treatable.

Surgical Rx: cardiac transplantation provides median 10 yrs survival & is effective palliation in appropriately selected individuals.

Prognosis

In the absence of a specific remediable aetiology (e.g. peripartum or alcoholic cardiomyo-pathy), the overall outcome is poor. The majority of pts have downhill course & partic-ularly those 〉55 yrs, die within 3 yrs of onset of symptoms. The 5 yrs survival rate of pts diagnosed é HF is 50%. The blacks are more likely to suffer from progressive heart failure & death than whites.

HYPERTROPHIC CARDIOMYOPATHY

HCM is characterized by LVH (myocardial thickness 〉1.5 cm), typically of none dilated chamber éout obvious causes. Other aetiologies of LVH, as long-standing hypertension & AS need to be excluded before one can diagnose HCM.

Genetic predisposition

HCM is the most common genetic CV disease. 50% of cases have +ve family history é autosomal dominant transmission. The prevalence in the general adult population for

people é phenotypic evidence of HCM is estimated at 1 per 500.

Pathophysiology

Generally, ventricular hypertrophy involves the proximal portion of the interventricular septum. As the septum thickens, it may narrow the outflow tract. In addition, systolic anterior motion of the mitral valve may occur & result in LV outflow tract obstruction & mitral regurgitation. When systolic anterior motion occurs, the mitral valve leaflets are pulled or dragged anteriorly toward the ventricular septum, producing the obstruction. Consequently, the LV has to generate much higher pressures to overcome the out flow obstruction & to pump blood to systemic circulation. Premature closure of the aortic valve may occur & is caused by the decline in pressure distal to the LV outflow obstruction.

Clinical features

Clinical course is variable. Most pts are asymptomatic. The most common symptom of HCM is dyspnea on exertion. The pt may also complain of chest pain é exertion, syncope or near syncope, or palpitations. CHF & AF along é their accompanying symptoms may be part of the natural history of HCM. Unfortunately, the first clinical manifestation may be sudden cardiac death, frequently occurring in young children/adults, often during or after physical exertion.

Physical examination

Unless CHF has developed, the **lungs are usually clear & JVP is normal. Bisferiens pulse**: rapidly rising carotid pulse followed by collapse in the pulse & then a 2ry rise. **Point of maximal impulse:** may be double or triple & sustained. **S4 gallop:** may be present. **Systolic murmur:** the classic finding for HCM is crescendo-decrescendo systolic murmur along the LSB that ⇧ é the valsalva manoeuvre. In young adults, HCM is the most common aetiology for sudden death.

Diagnosis

- **CXR:** may suggest LVH but will often be normal because hypertrophy in HCM involves the ventricular septum
- **ECG*:*** often shows LVH & occasionally have a pseudo infarct pattern. Left atrial abnormality may be present if the pt has had long-standing MR from systolic anterior motion of the mitral valve. AF may be present as a complications
- **ECHO:** is the gold standard for diagnosis. On transthoracic ECHO, the clinician should note the thickness of the septum; location & pattern of hypertrophy, degree of LV outflow tract obstruction, presence of systolic anterior motion of the mitral valve, presence of premature closure of the aortic valve & any change in severity associate é Amylnitrite.

Treatment

Medical Rx: competitive sport & strenuous exercise should avoided. Dehydration should avoided & diuretics should be used é caution. ß-blockers are considered as first line of Rx, by decreasing contractile force, ß-blockers ⇩ outflow gradient & ⇩ O_2 demand. ß-blockers also lengthen diastolic filling by slowing the heart rates. They help to control chest pain. Ca.Ch.Bl. is the 2nd line of Rx including Verapamil & Diltiazem can be used. But Nifedipine, Amlodipine & Felodipine should be avoided because they cause peripheral vaso-dilatation, ẃ may result in ⇩ LV filling & worsening of symptoms of outflow tract obstruction. AF is common complication of HCM. Rx of persistent AF in HCM includes anticoagulation & rate control é ß-blockers. Digoxin should be avoided in HCM pts, particularly in those é resting or latent obstruction, because of its +ve inotropic effect. Pts é HCM should receive prophylactic antibiotics for endocarditis before dental or invasive procedures. The turbulent flow through the LV-outflow tract striking the aortic valve as well as mitral regurge from systolic anterior motion of the mitral valve predispose to endocarditis.

Surgical Rx: septal myomectomy/myotomy may cause lasting symptomatic relief in 3/4 of severely symptomatic pts. Alcohol ablation: Ethanol injection into septal artery has reported to ⇩ obstruction.

Prevention: the first degree relatives of pt should be screened for ECHO.

RESTRICTIVE CARDIOMYOPATHY

Disease of myocardium, characterized by restrictive filling & ⇩ diastolic volume of either or both ventricles é normal or near-normal systolic function.

Pathophysiology

These conditions result in impaired ventricular filling & primarily diastolic HF. They present é clinical HF syndrome, that is frequently indistinguishable from that caused by systolic dysfunction. AF is poorly tolerated. It simulates other right side HF like cor-pulmonale & constricted pericarditis.

Clinical features

Exercise intolerance & dyspnoea are the prominent symptoms. Peripheral oedema é predominant ascites. Enlarged tender & pulsatile liver. ⇧ JVP ẃ does not fall normally during inspiration (Kussmaul sign). Ht sounds may be distant but apical impulse is easily palpable unlike in constrictive type.

Differential diagnosis: very similar to constrictive pericarditis.

Diagnostic work up

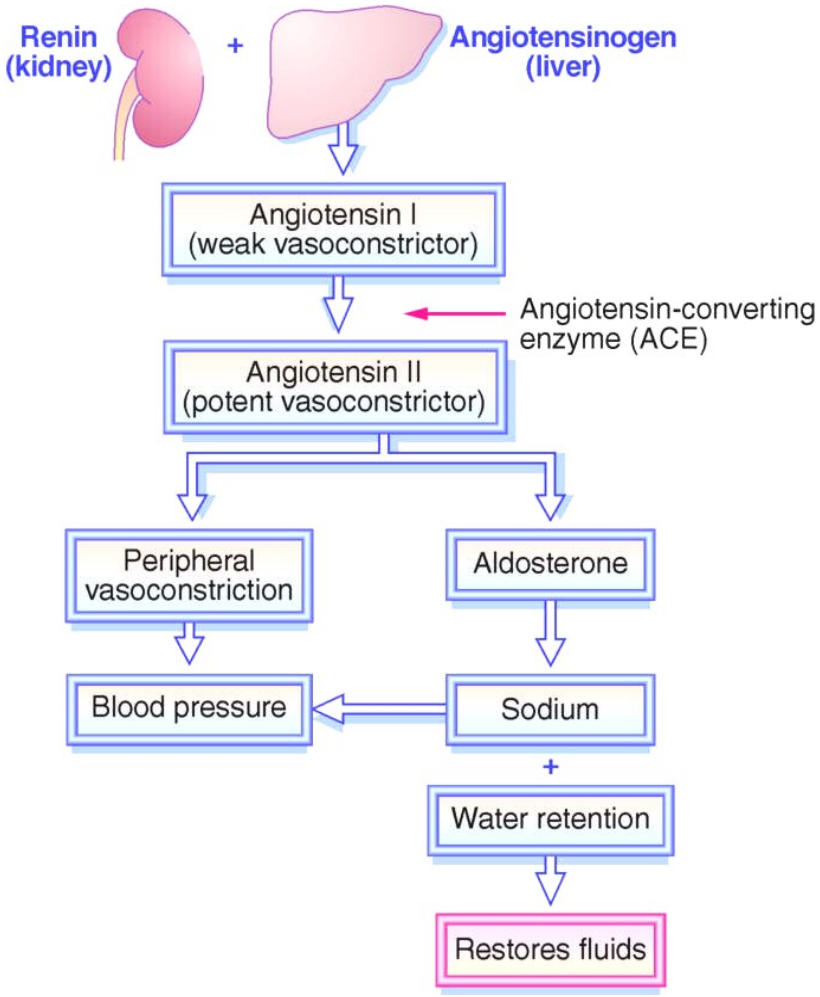
- **CXR:** mild cardiac enlargement.
- **ECG:** low voltage & conduction defects
- **ECHO:** ⇧ LV wall-thickness, ⇩ systolic function.

HYPERTENSION

Renin-Angiotensin-Aldosterone system

The kidney & blood vessels strive to regulate & maintain a normal BP, kidney regulate BP via the renin angiotensin system. Renin secreted from renal cells (jucsta glomerular apparatus) act on Angiotensinogen (produced by liver) changing it to Angiotensin I. In the lung Angiotensin I acted upon by angiotensin converting enzyme to form Angiotensin II ẃ is a potent vasoconstrictor & stimulant to adrenal gland to secrete Aldosterone. The aldosterone is steroid hormone (mineralocorticoid) secreted by zona glomerulosa of adrenal cortex, is under control of renin-angiotensin-aldosterone system & K^+ level in blood, also is under control of ACTH & the stretch receptors located in the atria of heart & the baroreceptors located in the large blood vessels. The aldosterone act on the renal tubules causing Na^+ & H_2O retention + ⇧ K^+ excretion in urine. Atrial Natriuretic Peptide is powerful vasodilator, secreted by atrial myocytes in response to ⇧ BP, act by dilating the afferent glomerular arteriole & constrict efferent glomerular arteriole so it cause ⇩ Na reabsorption in distal convoluted tubules & in the collecting duct, in addition to inhibition of renin secretion & ⇩ aldosterone secretion & inhibition of the effect of catecholamines in blood vessels causing relaxation of vascular smooth muscles in arterioles & venules (the opposite action of aldosterone). Hypertension is defined as arterial BP that exceeds 140/90 mmHg at several deter- minations. This is an arbitrary definition because a diastolic pressure of even 85 mmHg may be associated é ⇧ cardiovascular morbidity &mortality. Hypertension is one of the most common diseases affecting humans throughout the world. Because of the associated morbidity & mortality & the cost to society, hypertensi on is an important public health challenge. It is easily detectable, usually easily treatable & often leads to lethal complications if left untreated.

Hypertension is the most important modifiable risk factor for CHD, stroke, CHF, end-stage renal disease & peripheral vascular disease. Therefore, health care professionals must not only identify & treat pts é hypertension but also promote a healthy life-style & preventive strategies to ⇩ its prevalence in the general population.



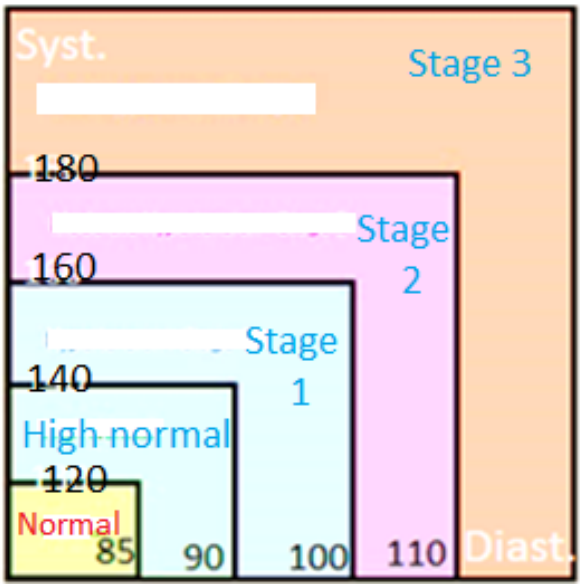
Epidemiology

Overall, 20% of the world's adults are estimated to have hypertension in excess of 140/90 mmHg. Some studies in developed countries show 50% of population have hypertension. The prevalence dramatically ⇧ in pts > 60 yrs. Prevalence is higher among blacks than whites. The age-adjusted prevalence of hypertension is slightly higher in men than in women. The prevalence in women is closely related to age, é substantial ⇧ occurring after age 50. This may be related to hormonal changes associated é menopause.

Classification

Because the risk to an individual pt may correlate é the severity of hypertension, a classification system is essential for making decision about aggressiveness of Rx or therapeutic interventions. When systolic & diastolic BP levels fall into different categories, the higher category should be selected to classify the individual's BP status e.g. 160/92 mmHg should be classified as stage “II” hypertension & 174/120 mmHg should be classified as stage “III” hypertension. Isolated systolic hypertension defined as systolic BP ≥ 140 mmHg & diastolic BP ˂90 mmHg & staged approximately (e.g 170/82 mmHg is defined as stage “II” isolated systolic hypertension). In addition to classifying stages of hypertension on the basis of average BP levels, clinicians should specify the presence or absence of target organ damage & additional risk factors. This specificity is important for risk classification & Rx. Optimal BP é respect to cardiovascular risk is ≤ 120/80 mmHg. Hypertension should be diagnosed based on the average of 2 or more readings taken at 2 or more visits after an initial screen.

	Blood Pressure mmHg		
Category	Systolic		Diastolic
Optimal	<120	&	< 80
Normal	<130	&	< 85
High-normal	130–139	or	85–89
Hypertension stages			
*Stage 1	140–159	or	90–99
*Stage 2	160–179	or	100–109
*Stage 3	>180	or	N > 110



Etiologic classification

Hypertension may be classified as either essential or secondary.

(I) Essential hypertension (90-95%): is diagnosed in individuals in whom generalized or functional abnormalities may be the cause of hypertension but no specific secondary causes are identified. The pathophysiology of essential hypertension is multifactorial & highly complex. A number of factors modulate the BP, including; humeral mediators, vascular reactivity, circulating blood volume, vascular calibre, blood viscosity, COP, blood vessel elasticity & neural stimulation. Contributing factors for essential hypertension include the following:-

- ***Genetic predisposition:*** the exact mechanism not established.
- ***Environment***: number of factors implicated as dietary salt intake & salt sensitivity, obesity, occupation, family size & crowding.
- ***Pregnancy-induced hypertension:*** toxemia of pregnancy.

(II) Secondary hypertension (5-10%): 2ry to an identifiable disorder include:-

- Renal (2.5-6%): variety of renal diseases may be accompanied by hypertension; Renal parenchymal disease: CRF, chronic pyelonephritis, acute & chronic glomerulonephritis, polycystic kidney, renin-producing tumour, Hyperuricaemia.
- Renovascular hypertension (0.2-4%) including; hyperlipidaemia, coarctation of aorta, renal artery stenosis, vasculitis & collagen vascular disease.
- Endocrinal (1-2%): DM. Oral Contraceptives. Adrenocortical hypertension- Primary Aldosteronism, Conn`s syndrome, Cushing syndrome, Congenital Adrenal Hyperplasia, Pheochromocytoma. Acromegaly. Myxoedema. Thyrotoxicosis.
- Neurogenic: psychogenic, ⇧ ICP, acute spinal cord section.
- Drugs & Toxins: alcohol & adrenergic medications.

Predisposing factors

- Strong F/H of hypertension.
- Age: secondary hypertension often develops before the age of 35 or after 55 yrs.
- Associated CV risk factors as; cigarette smoking, lipid abnormality or hypercholesterolemia, DM, family history of early deaths due to CV diseases & alcoholism.

Prevalence

Age (years)	Hypertension
18-29	4%
30-39	11%
40-49	21%
50-59	44%
60-69	54%
70+	64%

Effects of hypertension

Pt é hypertension die prematurely, the most common cause of death is heart disease, stroke & renal failure also frequent, particularly in pt é retinopathy.

1. Effects on heart

- LVH as a compensatory mechanism.
- Coronary artery disease/ IHD as; angina pectoris or MI which may lead to HF.

2. Neurologic effects

- Retinal changes: exudates: hard & soft exudates. Hge include; dot & bloat Hge. Thickening of arterioles: copper wiring/silver wiring. Abnormalities on arteriolovenular crossings, or papilledema.
- CNS dysfunction: cerebrovascular disease: TIAs; episodic dizziness, unilateral blindness, hemiparesis. Stroke either; ischemic due to atherosclerosis of cerebral vessels, or hemorrhagic stroke as a result of ⇧ arterial pressure & formation of vascular microaneurysms.

Hypertensive encephalopathy is another effect of hypertension is consists of severe hyprtension, altered state of consciousness, ⇧ ICP é papilledema & seizure. The focal neurologic deficits are uncommon.

3. Effects on the kidneys

Arteriolosclerosis of the afferent & efferent arterioles & the glomerular capillary tuft impairs renal function. Pt may have proteinuria & microscopic hematuria & later on CRF.

Risk factors for an adverse prognosis in hypertension

•Black race. •Youth. •Male sex. •Smoking. •Obesity. •DM. •Hypercholesterolem-ia.

•Excess alcohol intake. •Evidence of end organ damage.

Approach to a pt é hypertension

Hypertension is confirmed after an elevated BP ≥ 140/90 mmHg, properly measured, has been documented on at least 3 separate occasions (based on the average of 2 or more readings taken at each of 2 or more visits after initial screening).

An accurate measurement of BP is the key to diagnosis

Several determinations should make over period of several wks. At any given visit, an average of 3 BP readings taken 2 min apart using a mercury manometer is preferable. BP should be measured in both the supine &sitting positions, auscultating é the bell of the stethoscope. On the 1st visit, BP should be checked in both arms &in one leg to avoid missing coarctation of aorta or subclavian artery stenosis. The improper cuff size may influence BP measurement, wider cuffs preferable, particularly if pts arm circumference ›30cm. Pt should rest quietly at least 5 min before measurement. Although somewhat controversial, the common practice is to document phase V (disappearance of all sounds) of Korotkoff sounds as diastolic BP.

Practical points

•Normally there is diurnal variation of BP. We may hear 4th HS in hypertension.

•Isolated systolic hypertension seen in elderly (atherosclerosis).

•Hypertension may associated é steroids, NSAID, cough syrup, contraceptive pills, erythropoietin, liquorice ẃ contain natural mineralocorticoids causing Na & H_2O retention, or vitamins especially Vit B.

•White coat hypertension (⇧ BP of pt during doctor examination).

•Reduction of BP by 5-6 mmHg ⇩ the risk of stroke by 40% & the risk of coronary heart diseases by 15-20%.

•Most hypertensive pts will require combination of antihypertensive drugs to achieve recommended target of BP < 140/80.

•Hypertension in neonate occur in 2% of admission to NNICU, BP is usually 85 /60 mmHg. In children, BP calculated as follow: SBP = Age in yrs X 2 + 80. & DBP = 2/3 of the SBP. Causes of hypertension in infants, children include: IC He, Neuroblastoma, Congenital adrenal hyperplasia, Congenital anomalies kidnyes, Coarctation of aorta, Renal vein thrombosis, & Vesicoureteric reflux.

Patient evaluation

In evaluating a pt é hypertension the initial history, physical examination & laboratory should be directed at:-

(1) Establishing pre-treatment base line hypertension.

(2) Identifying correctable secondary cause of hypertension.

(3) Determining if target organ damage is present: pts may have undiagnosed hypertension for yrs éout having their BP checked. Therefore, a search for end organ damage should be made through proper history, physical exam & investigations.

(4) Determining whether other CV risk factors are present.

(5) Assessing factors ẃ may influence the type of Rx or changed adversely by Rx.

Clinical symptoms & history

Most pts é hypertension has no specific symptoms & are identified only in the course of physical exam. If pt develops symptoms, this may be attributable to: elevated BP itself, or due to the end organ damage associated é hypertension, or the underlying secondary disease. Some of the symptoms may be:-

*Headache: though popularly considered symptom of high BP, it is a characteristic of only severe hypertension. Such headaches are localized to the occipital region & present when the pt awakens in the morning but subsides spontaneously after several hrs. *Dizziness.

*Palpitation.

*Easy Fatigability.

*Impotence.

*Symptoms referable to vascular or target organ damage: include; Epistaxis, Hematuria, blurring of vision. TIA, episodes of weakness, dizziness or stroke (hemorrhagic or ischemic), angina, MI, pain due to dissecting aorta.

*__***Symptoms/history suggesting underlying disease:***__ include:-

•History of known renal disease, abdominal masses, anemia.

•History of repeated UTI may suggest chronic pyelonephritis.

•History of sweating, labile hypertension, headache, nervousness, postural dizziness, palpitations & wt loss may suggest pheochromocytoma.

•History of polyuria, polydipsia & muscle weakness may be suggest hypokalemia associated é aldosteronism.

•History of wt gain, emotional labiality may suggest Cushing syndrome.

•History of cold/heat tolerance, sweating, lack of energy, bradycardia or tachycardia may indicate hypo or hyperthyroidism.

•History of intake of contraceptives pills, liquorice & sympathomimetics should be looked for. Obtain a history of over-the-counter medication use or unsuccessful antihypertensive Rx trials.

Physical examination

Compare BP & pulse in the 2 upper extremities & in supine & standing position & the ⇧ in DBP when pt goes from supine to standing.

General appearance: round face & truncal obesity suggests Cushing syndrome.

Proper measurement of BP: a rise in DBP when the pt goes from supine to standing position is compatible mostly é essential hypertension while ⇩ BP in the absence of antihypertensive medications suggests 2ry hypertension.

Funduscopic evaluation of eye: for evidence of hypertensive retinopathy. Flame-shaped Haemorrhage, cotton wool exudates, papilledema & other neurologic signs raises the possibility of ⇧ intra cranial pressure.-

Palpation of all peripheral pulses should be performed: mainly palpation & auscultation of carotid arteries. Femoral pulse should be felt & compared é radial pulse. Radial femoral delay suggests COA.

Careful cardiac examination: to evaluate signs of LVH. These include displacement of apex, a sustained & enlarged apical impulse & presence of S4, occasionally, a tambour S2 is heard é aortic root dilatation.

Abdominal examination: look for renal artery bruit over the upper abdomen; the presence of a unilateral bruit é both a systolic & diastolic component suggests renal artery stenosis. Palpate for an abdominal aneurysm, enlarged kidneys of polycystic kidney.

Diagnostic workup

Laboratory investigations: unless a 2ry cause for hypertension is suspected, only the following routine laboratory studies should be performed including:-

- CBC, Hct.
- Urine analysis including microscopy, protein, blood, sugar.
- FBS, 2HPPBS • Electrolytes (K^+, Na^+).
- Lipid profile (cholesterol, LDL, HDL, triglycerides).
- Creatinine & Uric acid
- **ECG & Imaging studies:** ECHO to detect LVH.

Special studies: requested only when 2ry hypertension strongly suspected.

- U/S & doppler flow study for renovascular disease.
- 24 hrs urine assay of metanephrines & catecholamine for pheochromocytoma.
- Overnight dexamethasone suppression test or 24hr urine cortisol for Cushing sy.
- Plasma aldosterone for 1ry aldosteronism.
- Thyroid function (TSH, T3, T4) for thyrotoxicosis/Myxoedema.

Complications of Hypertension

- Hypertensive Cardiomyopathy: HF, LVF (congested lung, pt unable lie flat on bed) .RVF (enlarged congested liver, ⇧ of JVP, oedema of lower limbs & sacrum), MI.
- Hypertensive Nephropathy: chronic renal failure, in diabetic pt é hypertension, the early sign of development of diabetic nephropathy is the presence of microa-lbuminurea.
- Hypertensive Encephalopathy: confusion, headache, convulsion), CVA (stroke).
- Hypertensive Retinopathy.

Management: indication for Rx are; *SBP >140 & DBP >90 mmHg repeatedly.

*Isolated systolic hypertension SBP >160 & DBP <89 mmHg, if pt is >65 yrs.

General measures

***Sodium restriction:** intake **<**100 mmol/day (2.4 gm Na or 6 gm of Nacl).

***Lifestyle modifications:** weight reduction in obese pt. Limitation of alcohol intake as alcohol potentiates the action of catecholamines & may exacerbate hypertension. Regular physical exercise: ⇧ aerobic activity (30-45 minutes most days of the wk). Stop smoking. The DASH diet (Dietary Approaches to Stop Hypertension) include; adequate intake of dietary K, Ca & Mg (healthy diet like fruits, vegetables) & reducing the intake of dietary saturated fat & cholesterol.

Medications

Diuretics: also called water pills, often the 1st line drugs, reduce ECF volume. Act by flushing of excess H_2O & Na from the body, thus lowering the BP, may be enough along é life style changes to control BP in the start, include:-

Thiazide diuretics: are more effective antihypertensive agents than loop diuretics. Block Na & Cl reabsorption predominantly in the distal convoluted tubules,⇧ urine excretion of Na, Cl, K & Mg. **Chloro/Hydrochlorothiazide** 25 mg PO daily & may be ⇧ gradually. Side effects: Hyponatraemia, hypokalemia, hypomagnesaemia, Hypercalcemia, hyperuricemia, hyperglycemia, weakness, muscle cramps, impotence, hype-rlipidaemia (⇧ LDL & trigly-ceride), thiazide induced pancreatitis. Thiazide contraindicated in pt é Gout.

Loop diuretics: block Na, Cl & K reabsorption in the thick ascending loop of Henle & the most effective agent in pt. é renal insufficiency (Cr. **>**2.5 mg). **Furosemide** 20, 40 mg tab/amp, 20-320 mg/day. Side effects: hypo k/Ca/Mg, & ototoxicity.

Potassium-sparing diuretics: is competitive inhibitor of aldosterone on kidney, may be used in 1ry hyperaldosteronism (as additional therapy in combination é thiazide diuretics). **Spironolactone** 25, 50, 100 mg tab, 25-100 mg/day. Side effects: hyperkalemia,

gynecomastia in males & breast tenderness in females.

Angiotensin Converting Enzyme Inhibitor

Block the production of Angiotensin II by preventing conversion of Angiotensin I to Angiotensin II. By doing so ACEI reduce peripheral resistance. In addition they reduce Aldosterone production, reducing the retention of Na^+ & ⇧ GFR. Effective in pt é hypertension associated é HF, kidney disease, DM, or CT diseases.

Captopril 12.5-75 mg PO BID.

Enalapril: 2.5-40 mg daily. Side effects include; cough, leucopenia, angioedema, hyperkalemia, hyponatraemia, taste disturbance & first dose hypotension. Contraindicated in bilateral renal artery stenosis & renal failure.

Angiotensin II Receptor Blockers (ARBS)

Prevent Angiotensin II (ẃ is potent vasoconstrictor causing smooth muscles surrounding blood vessels to contract) from binding to angiotensin receptors, block vasoconstriction & block release of aldosterone. Used in hypertensive pt é renal impairment, diabetic nephropathy, AF, HF & those unable to tolerate ACEIs as result of marked cough or development of angioedema.

Irbesartan tab 1500 mg/day. **Losartan** tab 25-50 mg once or twice daily.

Side effects include: hyperkalemia, headache, cough, hypotension, angioedema, dizziness, allergic reaction. Are contraindicated during pregnancy.

Action of ACEI & ARBS: vasodilatation, ⇩ BP, ⇩ peripheral resistance, diuresis, no changes in HR, no reduction of COP.

β-Blockers

The β-receptors are found on cells of the heart muscles, arteries, smooth muscle, airway, kidneys & other tissues that are part of the sympathetic nervous system. β-receptors blo-

ckers block the action of catecholamines epinephrine (adrenaline) & norepinephrine (noradrenalin) in particular, on β-adrenergic receptors by competitive inhibition. There are 3 types of β-receptors:-

- β1-adrenergic receptors: are located mainly in heart & kidneys.
- β2-adrenergic receptors: are located mainly in lungs, GIT, liver, uterus, vascular smooth muscles & skeletal muscles.
- β3 adrenergic receptors: are located in the fat cells.

The β-blockers used mainly for the Rx of cardiac arrhythmia, protecting heart from 2nd heart attack, angina & hypertension. The **β1-Receptor Blocker** is cardioselective preferred,⇩ HR, COP & contractility of heart, also prolong P-R interval, ⇧ prostaglandine ẃ is potent vasodilator & ⇩ renin release from the kidneys & cause no bronchospasm compared by the non-selective β-blocker ẃ act on both β_1 (he-art) & β_2 (bronchial) receptors. **Propranolol** is non-selective β-Blocker, 20 mg PO/ day to maximum of 120 mg PO 4 X/day. **Metoprolol** is selective β-Blocker, 25-150 mg PO BID or **Atenolol** (Tenormin/ Blokium tablet), 25-100mg PO/day.

The side effects of β-blockers: bradycardia, worsening of HF, AV block, dry mouth, depression, ⇧ serum lipids, hypoglycemia, hyperkalemia, bronchospasm, aggravate bronchial asthma & COPD, insomnia, night mares, hallucination, impotence & psychosis. The contraindications for β-blockers are; bronchial asthma, or peripheral vascular disease.

Calcium channel blockers

Inhibit inward movement of calcium ions through the slow channels of active me-mbrane in myocardial & vascular smooth muscle cells, causing -ve inotropic effect in the heart, prolong depolarization, ⇩ myocardial O_2 consumption, coronary & peripheral vasodilatation (smooth muscle cells relaxation). Used in elderly pt é hypertension, angina or é

cardiac arrhythmia. The Ca Ch BLs pharmacodynamics include: ⇩ BP, ⇩ HR, ⇩ of stroke volume, ⇩ COP, ⇩ total peripheral resistance.

The Ca Ch Bls include 2 groups:-

① **Dihydropyridines** (vascular effect): **Nifedipine** 30-90 mg PO/ day. **Amlodipine** 2.5-10 mg PO daily. Contraindicated é heart block or HF.

② **Non Dihydropyridines** (cardiac effect): **Diltiazem & Verapamil**. **Isoptin** 80, 120 mg tab, 80-480 mg/day, have cardio depressant effect & their use may be problematic é CHF.

Side effects of Ca Ch BLs: constipation, headache, peripheral oedema, palpitation, dizzyness, fatigue, visual disturbances & mental depression.

Centrally acting agents

These agents inhibit sympathetic out flow from the CNS. Are vasodilators: dilate arterioles & arteries, ⇩ peripheral vascular resistance ẃ in turn reduces high BP. ***α* blockers;** block the effect of sympathetic nerves on blood vessels by binding to α-adrenoreceptors located in the vascular smooth muscle, most of these drugs act as competitive antagonists to the binding of norepinephrine that is released by sympathetic nerve synapsing in smooth muscle. Therefore sometimes referred to as sympatholytic. The side effects of α blockers include; weakness, fainting when standing up suddenly, nasal congestion & headache.

Prazosin 1, 2 mg tab, dosage range 1-20 mg/day.

Methyldopa 250-1000 mg PO BID, TID or QID. Side effects: postural hypotension, depression, gynecomastia.

Hydralazine 10-75 mg PO QID, 10-50 mg IV. Side effects: headache, SLE like sy.

Minoxidil 2.5-4.0 mg PO BID. Side effects: orthostatic hypotension.

Stepwise prescription of anti-hypertensive medication

Diuretics are often prefered as first line drugs. They may be effective alone in mild hypertension. However most of the time they are used in combination é other drugs as β-blockers, ACEI or Ca Ch BL. Hydrochlorothiazide will potentiate the activity of a number of antihypertensive drugs, particularly ACEI. Such combination has an additive effect, controlling BP in up to 85% of pts. The dosage of antihypertensive drugs should be escalated till BP is well controlled. If BP still uncontrolled consider using multiple drugs acting at different sites & have additive effect. Most drug combinations, using agents that act by different mechanisms, have an additive effect. The combination of Ca Ch Bls é an ACEIs has additive effects. Some combinations may not be additive, including β-blockers & ACEIs or βblocker & α1 blocker or α 2 stimulant. Some combinations may have additive adverse effects; these include β-blocker combined é Verapamil or Diltiazem (Ca Ch Bl), ẃ leads to cardiac depression bradycardia, or AV block. If the BP is still resistant to Rx add direct vasodilators.

For summary of the stepwise prescription:-

Elderly hypertensive (> 60 yrs): generally pt have ⇧ vascular resistance, ⇩ plasma renin & greater LVH than younger pt, so use diuretics as first line, or Ca Ch Bls ẃ ⇩ the peripheral resistance & has no adverse effects on lipid level.

Diabetic hypertensive pt: usually have diabetic nephropathy, proteinurea & renal insufficiency, use ACEIs or ARBs as first line, they shown to ⇩ rate of progression to end stage renal disease & ⇩ retinal complications.

Hypertensive é chronic renal insufficiency: diuretics used to deal é the Na & H_2O retention, loop diuretics are the most effective class, ACEIs are shown to slow the rate of deterioration of renal function.

Hypertensive é coronary artery disease: pt is at high risk for development of MI & angina. β-blockers used as first line of Rx. ACEIs or ARBs are also useful in such pt é LV dysfunction.

Hypertensive é HF: ACEIs or ARBs is beneficial in ⇩ the mortality. β-Blockers & Aldactone also improve outcome. Don`t use NHPor α-Blockers.

Hypertensive é Ht block: never use β-Blockers or NHP.

Hypertensive é asthma or COPD: never use β-Blockers.

Hypertensive é Gout: never use diuretics as it worsen the condition, ARBs are good choice to reduce serum urates.

Hypertensive é benign prostatic hyperplasia: α- blockers is good choice.

***Thiazides:** preferred therapy for uncomplicated hypertension, or é systolic hypertension in elderly people & older diabetic pt éout nephropathy.

***ACEIs:** should be the initial Rx in situations in ẃ hypertension is associated é CHF, or DM é proteinuria or post MI é systolic LV dysfunction, or in pt who develop persistent cough while on ACEIs Rx, an ARBs may be substituted, but these agents' efficacy in lowering cardiovascular mortality rates has not yet been proven.

***B-Blockers:** often preferred in post-MI & uncomplicated hypertension.

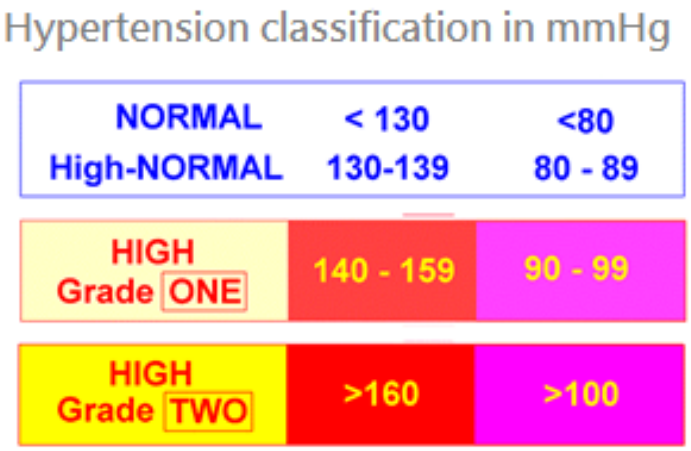
***Diuretic or long-acting Ca Ch Bls:** effective in elderly é isolated systolic hypertension.

***Ca.Ch.Bl.:** Nifedipine is preferred Rx for systolic hypertension & alternative therapy in uncomplicated hypertension.

***Central acting agents (e.g. M*ethyldopa**):** may be used as alternative Rx for uncomplicated hypertension.

Side effects of commonly used drugs

Diuretics	Hypokalaemia, hypomagnesaemia, ototoxicity, orthostatic hypotension, hyperuricemia
B- Blockers	Bradycardia, hypotension, AV block, dizziness, fatigue, depression, diarrhoea, nausea, vomiting, bronchospasm, hypoglycemia, hyperglycaemia
ACEIs	Angioedema, dry cough, hyperkalaemia, dizziness, hypotension, fatigue, syncope, rash, nausea, vomiting
ARBs	Orthostatic hypotension, diarrhoea, hyperkalaemia, dizziness, fatigue, myalgia, nasal congestion, insomnia, syncope
Ca Ch Bls	Bradycardia, hypotension, tachycardia, ventricular fibrillation, dizziness, fatigue, peripheral oedema, nausea, vomiting, constipation, anorexia, flushing, ⇧ liver enzymes, AV block
α- blockers	Orthostatic hypotension, dizziness, sinus bradycardia, vertigo, syncope, diarrhoea, fatigue, peripheral oedema, nausea, vomiting, priapism, impotence, floppy iris syndrome



HYPERTENSIVE CRISIS

Defined as severe hypertension characterized by DBP >130 mmHg. The BP elevation to such degree can cause vascular damage, encephalopathy, retinal Hge, renal damage & death. 1-2 % of the hypertensive population develop this complication. It is categorized into:-

(1) Hypertensive emergency: there is acute impairment of an organ system (CNS, CVS, Renal). In these conditions, BP should lowered aggressively over minutes.

(2) Hypertensive urgency: BP is high & there is potential risk but not yet caused acute end-organ damage. These pts require BP control over several days to wks.

Diagnosis: DBP of 130 mmHg, funduscopic finding of papilledema, change in neurologic & mental status & abnormal renal sediments are the hallmarks. Approach to pt é hypertensive crisis include; rapid assessment of the pt é brief history & targeted physical examination (of the CNS, CVS & Retina).

Laboratory investigations: ▲CBC. ▲Urine analysis. ▲Renal function test. ▲ECG.

Treatment "treats the pt not the number".

General measures: look if the pt is in stressful situation. Place pt in quiet room & re-evaluate after initial interview. Some pt's BP ⇩ below a critical level after relaxation.

Pharmacologic Rx: if pt has hypertensive emergency, lower the BP rapidly by 25% of the diastolic BP & not <95 mmHg. Use rapidly acting drug as IV Sodium Nitroprusside, Hydralazine, or sublingual Nifedipine.

Sodium Nitroprusside 0.5-8 ugm/kg/min IV infusion till BP is lowered to normal or

Hydralazine 10-20 mg IV stat to be repeated every 20-30 min or

Labetalol 2mg/min through continuous IV infusion.

As the pt BP stabilizes start long term oral medications.

ISCHEMIC HEART DISEASES

Myocardial ischemia occurs when the blood flow demands of the heart exceed the blood supplied by the coronary arteries.

Epidemiology

IHD is the leading cause of morbidity & mortality in developed countries & it incurs greater economic cost. The prevalence of IHD is on the rise in developing countries as there is change in the life style associated é urbanization including sedentary life style, smoking, obesity, high fat & energy diet & the associated ⇧ prevalence of DM. Larger ⇧ in prevalence of IHD throughout the world are projected & is likely to become the most common cause of death worldwide by year 2020.

Etiology/Pathophysiology

Myocardial ischemia reflects an imbalance between the myocardial O_2 supply & demand. Myocardial O_2 demand is mainly determined by heart rate, the force of ventricular contraction & ventricular wall tension, ẃ is proportional to the ventricular volume & pressure. Unless there is a proportionate rise in O_2 supply, conditions that ⇧ O_2 demand such as physical exertion result in ischemia. Atherosclerosis is another factor.

Risk factors

Age: the risk of CAD ⇧ progressively é age. The risk of death from coronary artery disease is 1.5/1000 individuals at age 50.

Gender: IHD is more prevalent in men than women. The difference is more marked in premenopausal women compared to men of similar age.

Lipid abnormalities: ⇧ serum LDL & ⇩ HDL é hypertriglyceridemia favors the deposition of lipids & cholesterol in atherosclerotic plaques (hypertriglyceridemia commonly is associated é DM).

Smoking: the smokers are 60% more likely to develop CAD than the non-smokers.

Hypertension: ⇧ the risk of CAD both in men & women. DM is associated é significant ⇧ in the risk of CAD.

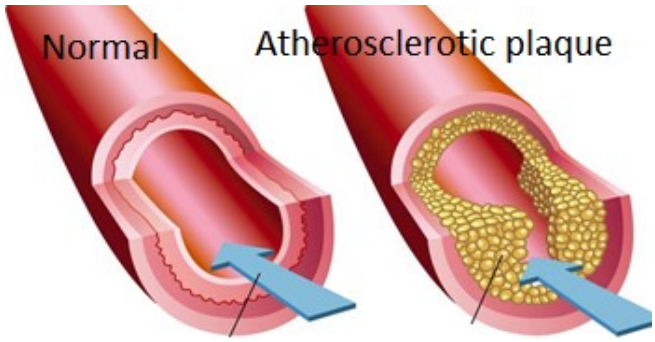
Family history: a familial predisposition to CAD exists.

Oral contraceptive pills: associated é ⇧ risk of CAD.

Other risk factors: gout & obesity.

Atherosclerosis

Is focal narrowing of arteries ẃ results from plaque formation. CAD & atherosclerosis risk factors such as hyperlipidemia, smoking, DM & hypertension apparently disrupt the normal functioning of the vascular endothelium. Plaques are formed as a result of:- intimal smooth muscle proliferation probably as a result of endothelial damage, or due to lipids (cholesterol esters) deposition at the center of plaque & also within smooth muscle cells. A fibrous cup made of connective tissue covers the plaque. As the stenotic lesions grow, perfusion pressure distal to the lesions ⇩; in response, coronary arterioles dilate to maintain adequate blood flow preventing ischemic symptoms at rest. During exertion the myocardial O_2 demand ⇧ ẃ could not be matched by the perfusion via narrowed coronary artery. The resulting myocardial ischemia results in chest pain, ẃ is relieved by taking rest. Sometimes atherosclerotic plaques may rapture & a fibrin thrombus is formed over the plaque ẃ completely blocks the narrowed coronary artery & result in MI.



ANGINA PECTORIS

Is a chest pain or pressure produced by myocardial ischemia. Often ppt by exertion or other factors that ⇧ myocardial O_2 demand (e.g. emotional stress, eating meal, sexual intercourse) may ppt angina. Chest pain in angina is squeezing in type or a feeling of pressure or tightness in the chest. Sometimes it can be burning in nature or felt as epigastric discomfort. The pain radiates to the left shoulder, left jaw, teeth or to the left arm & sometimes may radiate to the right arm. The pain in angina is often reproducible é the same degree of physical exertion. The symptom usually begins é low intensity, ⇧ over 2-3 min & often lasts ‹15 min. An episodes lasting ›30 min suggest MI may have occurred.

Types

Classic or excertional angina: pain ppt by ⇧ workload on the heart. May be caused by exercise, emotions, stress & cold exposure. Symptoms may remain "stable" for a number of yrs or progress in severity.

Silent ischemia: is particularly dangerous form of myocardial ischemia as there is a lack of clinical symptoms, i.e. ischemia éout angina. For every episode of symptomatic ischemia that the pt suffers, there are usually 4-5 episodes of silent (asymptomatic) ischemia. Can be detected by ECG including stress ECG or Holter monitor. Such episodes are less severe in nature & shorter in duration.

Unstable Angina: is angina that occurs at rest. Also referred to as "pre infarct angina" since it usually associated é extensive blockage of the coronary arteries. The coronary blood flow does not meet the needs of the heart even at rest. Unstable angina is progressive & may be ominous feature of imminent MI. So physicians & pts should be aware that close observation & intensive therapy are required. It represents a more serious situation than chronic stable angina.

Variant angina (vasospastic angina, prinzmetal's angina): this is type of angina resulting from transient coronary spasm, usually associated é fixed atherosclerotic lesion. The spasm produces total but transient coronary occlusion. Usually occurs at rest (often at night) & frequently complicated é ventricular arrhythmias.

Diagnosis

In pt presenting é history of recurrent chest pain, obtain detailed history including onset, quality, location, duration, radiation, ppt & relieving factors. Determine presence or absence of each of the 3 following symptom complex characteristics:-

① Substernal discomfort é characteristic quality & duration.

② Symptoms provoked by exertion or emotional stress.

③ Symptoms relieved by rest or nitroglycerine.

Based on the number of symptom complex present, angina can be classified as:-

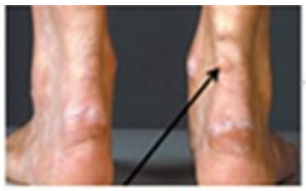
- ***Typical (definite) angina:*** all the above 3 characteristics are present.
- ***Atypical (probable) angina:*** only 2 of the above 3 characteristics are present.
- ***No cardiac chest pain:*** 1 or none of the 3 characteristics is present.

If the pt's symptoms are consistent é definite angina, sub classifies the angina as **stable** (unchanged for 2 or more months) or **unstable** (at rest, or new-onset, or increasing angina). In pt é definite or probable angina, ask about functional limitations. Ask about any episodes of dyspnea, palpitations or dizziness é/éout chest pain. If the pt has experienced any such episodes, ask about the same symptom variables applicable to typical (definite) angina. Assess potential risk factors for CAD, including those related to lifestyle, habits (smoking), medical history, family history, hormone therapy. Ask about history of symptoms such as excertional dyspnea, orthopnea & bilateral leg swelling,that suggest LV

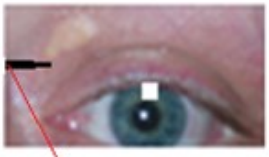
dysfunction or HF. Ask about nocturia. Ask about other potential causes of chest pain, especially if symptoms have changed or new symptom have arisen, or pt is at low risk for CAD.

Physical examination

In pt presenting é suspected stable angina (unchanged for 2 or more months), perform complete physical examination to help identify the cause of the chest pain & any combined disorder(s). Assess vital signs, especially for hypertension, tachycardia, bradycardia, arrhythmia & tachypnea. Closely examine the head & neck, especially for signs of anemia (mucous membrane pallor), thyroid disease (exophthalmos, thyromegaly), hypercholesterolemia (xanthelasma ẃ is lipid deposit in the skin of the eyelids) or atherosclerosis (carotid brut). Examine the lower extremities for dependent (ankle) oedema, tendinous xanthomas (lipid deposit), weak pulses, or cutaneous signs of ischemia or necrosis. Perform general & peripheral vascular examination to identify signs of generalized or peripheral atherosclerosis (e.g. inequality of BP in arms, diminished pedal pulse & abdominal aneurysm). Carefully examine the heart for evidence of hypertrophy, murmur & 3rd or 4th heart sounds. Examine lungs for rales & abnormal sounds



Tendinous xanthomas



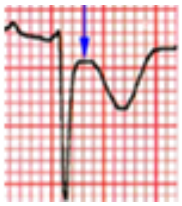
Xanthelasma

Diagnostic workup

ECG: if taken when pt is not in pain may be normal. The presence of new horizontal or down sloping of ST segment & new T-wave inversion are suggestive of myocardial ischemia. ST segment elevation associated é pain ẃ returns to normal as the pain wanes suggest variant angina. There are some things you should bear in mind before you can say if

the pt really has angina or not. You need to see T wave changes on 2 or more consecutive leads (not including AVR) for you to get worried about it. The other

thing you can see on angina ECG is that the T wave (the last wave) is upside down. T wave inversion or even flattening is another sign of angina, but it is not a good one. It could also signify an old or current heart attack (MI).



ST depression

But clue like this is useful when you become more experienced & you are trying to interpret more difficult ECG's. Remember that history & change on the ECG is more important than a ECG by itself. ECGs are not fool proof - If you convinced that the pt has angina from the history but the ECG is normal then you should still contact some one senior. ECGs can be normal when really something awful happening.

Stress ECG: recording ECG during exercise ⇧ the sensitivity & specificity of ECG. This helps to quantify the pt's exercise tolerance. Presence of new horizontal or down sloping of ST segment depression has sensitivity of 70% & specificity of 90%.

Holter monitoring: 24 hrs ambulatory ECG, for different types of angina.

Radiologic/Imaging: **CXR** in pt é CHF.

Stress radionuclide ventricuography, *Stress ECHO & Cardiac catheterization.*

Other laboratory tests: CBC, FBS & lipid profile.

Differential Diagnosis

Consider other causes of chest pain like; pleurisy, pneumonia, pericarditis, ischemia associated é AS or hypertrophic cardiomyopathy.

Treatment

Therapy for angina should be directed either towards reducing myocardial O_2 demand, or to compensate for impaired flow through diseased coronary arteries or ⇧ myocardial O_2 supply (blood flow).

Life style measures:

- Counsel pt about cessation of smoking.
- Diet: ('healthy' diet like low fat, low caloric diet é ⇧ habit of eating fruits & vegetables).
- Regular exercise.
- Wt reduction.

Organic nitrates: this class of drugs produce venodilatation & to lesser extent arteriolar dilatation. Dilate peripheral veins & so ⇩ preload. Dilate coronary arteries & so ⇧ myocardial blood flow. Dilate peripheral arteries & so ⇩ the afterload (afterload is the force that contracting heart must generate to eject blood, the afterload is affected by peripheral vascular resistance & BP). These effects of organic nitrates ⇩ the BP & cardiac size. You should instruct pt about how & when to use the short-acting nitrates (e.g. 0.4mg sublingual nitroglycerin) for the acute attacks, unless nitrates are contraindicated. Consider the long-acting nitrates in pt who have refractory chest pain despite maximal tolerated ß-blocker therapy, or in pt. who would benefit from after load reduction. To avoid nitrate tolerance in pt requiring long-acting nitrates, prescribe a 10-12 hrs nitrate-free period daily.

Nitroglycerine: 0.3-0.6 mg sublingual as soon as the pain starts or 5 min before a stressful activity.

Isosorbide dinitrate slow release: 10-60 mg PO TID or 2.5-10 mg sublingual/4-6 hrs. Major adverse effects of nitrates are headache, hypotension & tolerance.

β- Blockers: act by blocking myocardial β-adrenergic receptors. ⇩ HR & COP, also ⇩ myocardium workload (contractility). By doing so ⇩ cardiac O_2 demand.

Propranolol- is non-selective β1 & β2 blockers 20-80 mg PO BID-QID.

Metoprolol- is selective β1 blocker, 25-200 mg Po BID.

Atenolol- selective β1 blocker, 50-150 mg PO daily.

Consider the pt concomitant health problems when selecting a specific ß-Blocker.

β-Blockers are contraindicated in pt é asthma or severe CHF.

Side effects: bradycardia,⇩ COP & bronchoconstriction é the nonspecific drugs.

Ca Ch Bls.: act by ⇩ smooth muscle tone of coronary arteries, especially effective in preventing coronary spasm that cause variant angina. Also used for hypertension & arrhythmia. Mechanism of action is through blocking the calcium channels in vascular smooth muscles, dilate coronary arteries & ⇧myocardial blood flow, also dilate peripheral arteries & ⇩ the afterload. Consider Ca Ch Bls when ß-blockers are contraindicated or not tolerated. Consider using combination therapy cautiously (e.g. ß-blocker + Ca Ch Bl or nitrate) for pt who fail to respond adequately to monotherapy.

Nifedipine XL 30 mg PO daily.

Verapamil 180-240 mg daily.

Amlodipine 5-10 mg daily. Side effect: headache, hypotension, reflex tachycardia, risk of heart block& HF particularly é verapamil.

Antiplatelet agents: Aspirin prevent platelet aggregation, use for prophylaxis of blood clots particularly in unstable angina, small dose is recommended for pt é angina to prevent the occurrence of MI. 75-150 mg PO daily. Alternative is Plavix.

Lipid lowering drugs: generally, prescribe a lipid-lowering medication (Statins) & low-fat, low-cholesterol diet for pt é CAD & ⇧ LDL levels to achieve an LDL level of ˂100 mg/dl.

ACE inhibitor: may be beneficial in pt é significant CAD by angiography or for those é previous MI who have DM &/or LV dysfunction.

Treat comorbidities: that can provoke or exacerbate angina (e.g., hypertension, DM, hyperthyroidism, pulmonary disorders, anemia). Always refer pt presenting é new-onset angina, or rest angina, or increasing angina to an emergency department & hospitalize pt é clinical evidence of unstable angina or MI.

Coronary angioplasty: uses a balloon catheter to open occluded blood vessels. Performed under local anesthetic. Carries 5% mortality &high rate of vessel reocclusion. Use of stent in opened vessel ⇩ the rate of occlusion. Stent is effective during the first 3 hrs after occ-urrence of MI, new stents include; drug impregnated & self-absorbable one. Up to 6 or 8 stent may be used sometimes.

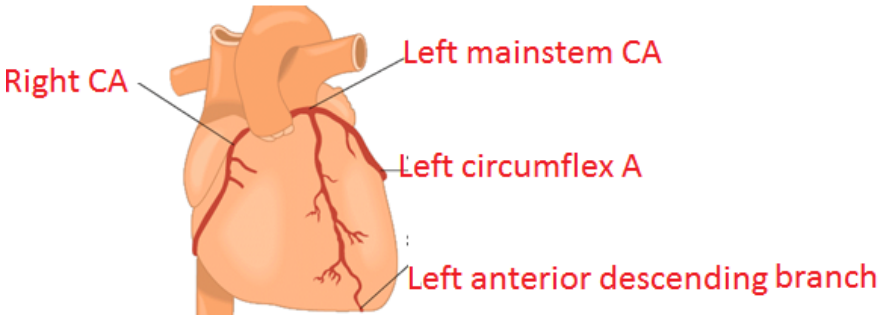
Coronary artery bypass: revascularization procedure in ẃ a blood vessel is taken from elsewhere in the body & surgically sutured around a blocked coronary artery. May involve multiple (1-5) vessels. Reocclusion of transplanted vessel is possible.

Prognosis: depends upon status of coronary arteries & LV function.

MYOCARDIAL INFARCTION

AMI or "heart attack" is an irreversible injury to & eventual death of myocardial tissue result from ischemia & hypoxia, is a leading killer of both men & women. Most heart attacks result from occlusion of coronary blood vessel by lipid deposit. These lipid deposits may accumulate to the point where they completely block a coronary vessel or, more commonly, accumulated lipid plaques may break off from the vascular endothelium & act as thrombus that blocks coronary artery at a narrower point downstream. Prolonged vasospasm might also ppt a AMI.

Coronary blood flow



The location of a MI will be largely determined by ẃ coronary blood vessel is occluded. The 2 main coronary arteries supplying the myocardium are:-

*left coronary artery: subdivides into left anterior descending & circumflex branch.

*Right coronary artery.

When myocardial blood supply is abruptly ⇩ or cut off to a region of the heart, a sequence of injurious events occur beginning é ischemia (inadequate tissue perfusion), followed by necrosis & eventual fibrosis if the blood supply is not restored in an appropriate period of time.

Types of Myocardial infarction

- Transmural: involves full thickness of ventricular wall, tend to have a greater effect on cardiac function & pumping ability.

- Subendocardial: involves inner 1/3 to 1/2 of the ventricular wall.

Symptoms

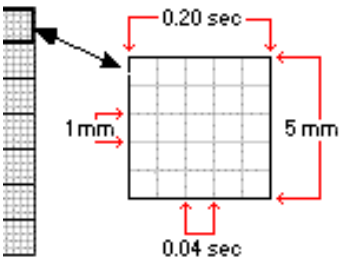
Chest pain: sever squeezing or crushing type of chest pain that lasts for **>** 30 min & not relived by rest or sublingual nitroglycerine. Pain radiates in a similar pattern to angina. Usually occurs when pt is at rest or involved in minimal activity. Emotional stress may also ppt AMI. Pt deny presence of a problem & try to find an explanation. Also significant % of AMI are "silent" é no symptoms & may be discovered on doing routine ECG.

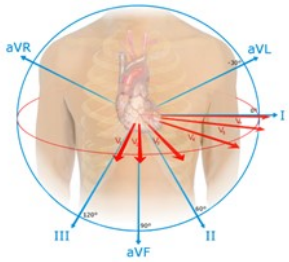
Other associated symptoms: Nausea. Vomiting. Excessive sweating. Shortness of breath. Anxiety & Sense of impending doom.

Physical examination

Pt is in pain &quite apprehensive often appears ashen. ⇩BP & ⇧HR, seen é extensive infarction, signs of CHF may be seen. A new murmur of MR may be present.

Diagnostic work up

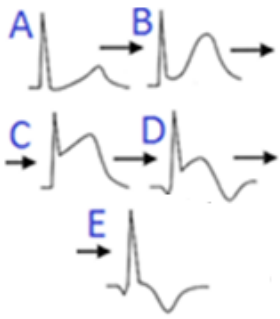




① ***ECG****:* diagnostic in ~85% of cases. To diagnose MI you need to go beyond looking at a rhythm strip & obtain a 12-Lead ECG. The 12-Lead ECG sees the heart from 12 different views. Therefore, the 12-Lead ECG helps you see what is happening in different portions of the heart.

Clue: look at AVL & AVF, if no changes of both ⇨ Anterior MI. If AVF show changes ⇨ Inferior MI. If AVL shows changes ⇨ Lateral or Anterolateral.

ECG changes over time é AMI occurs as follow:-



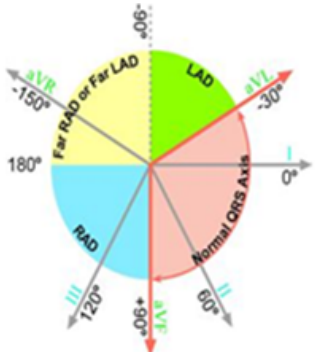
(A)Normal ECG

(B)Peaked T waves

(C)Marked ST elevation

(D) &(E) Ongoing infarction.

Determination of axis deviation



I	II	Axis
+	+	Normal
+	-	Left axis deviation
-	+	Right axis deviation
-	-	Right superior axis deviation

A quick way to determine the QRS axis is to look at the QRS complexes in leads I & II.

Normal axis deviation: the QRS axis falls between -30^0 ⇨ $+90^0$ because ventricular depolarization is leftward & downward.

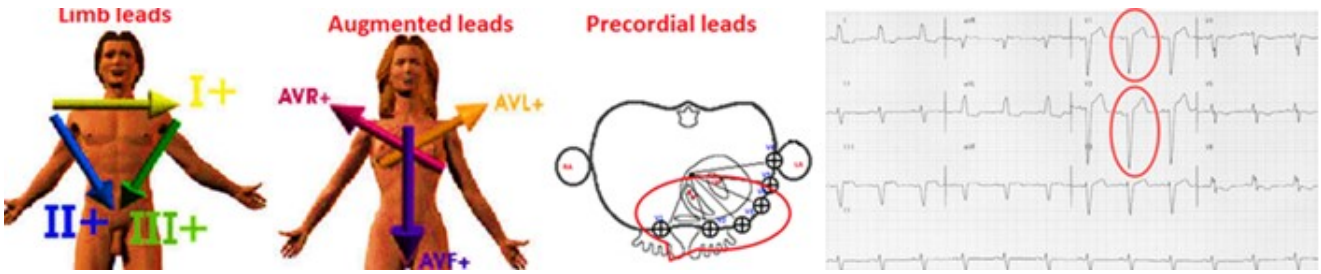
◍ LAD: occurs when axis falls between -30^0 - 90^0.

◍ RAD: occurs when axis falls between $+90^0$ ⇨ $+150^0$.

◍ RSAD: occurs when axis falls between $+150^0$ ⇨ -90^0.

ECG changes on MI:

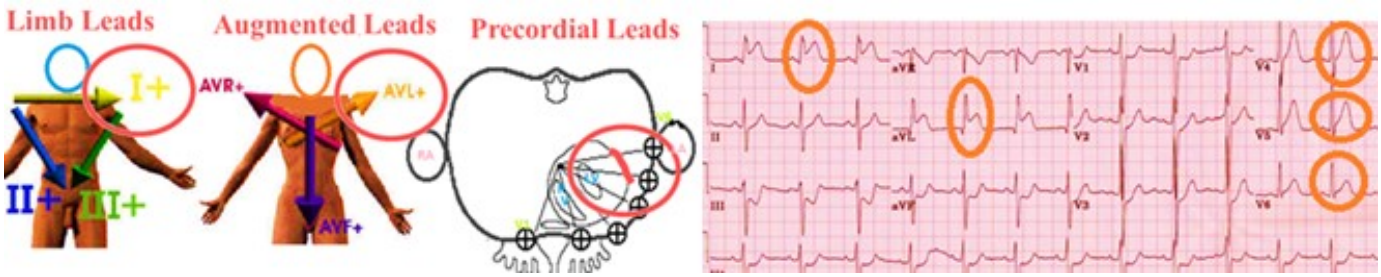
***Anterior MI: If you see changes in leads V1, V2, V3, V4 that are consistent é MI, you can conclude that it is an anterior wall MI.



Interpretation: Yes, this person is having MI involves the anterior wall. Note the ST eleva-

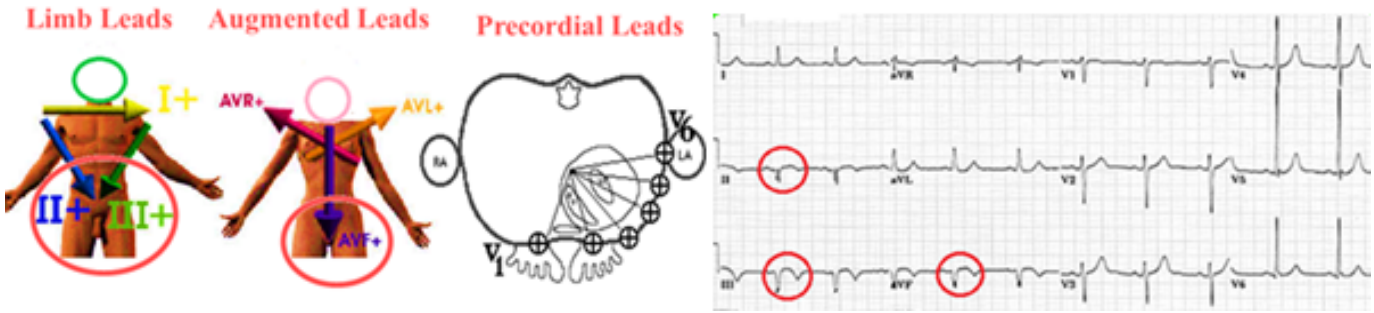
tion in leads V1, V2.

*** Lateral MI: If you see changes in leads: I, AVL, V4, V5,V6



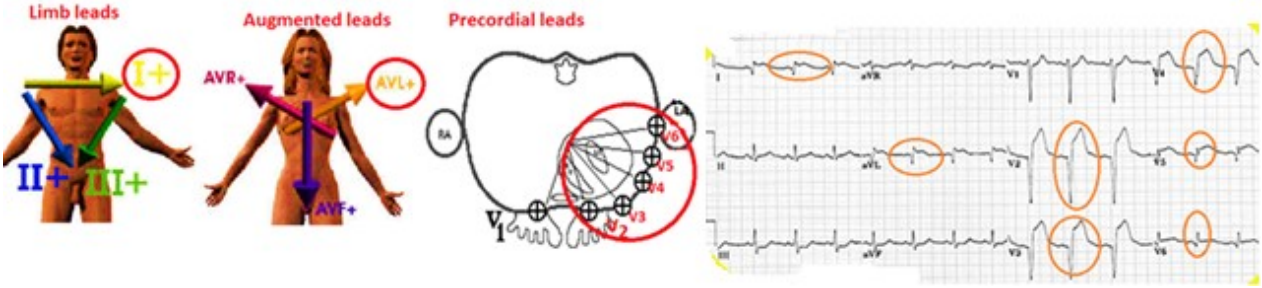
Interpretation: Yes, this person is having MI involves lateral wall (I, AVL, V4- V6)!

*** Inferior MI: If you see changes in leads: II, III, AVF



Interpretation: Yes, this person is having MI involves the inferior wall. Note the ST elevation in leads II, III, AVF.

*** Anterolateral MI: If you see changes in leads: I, AVL, V5, V6 + V2, V3, V4.



Interpretation: Yes, this person is having MI involves both the anterior wall (V2, V3, V4) & lateral wall (I, AVL, V5, V6)!

② ***Cardiac enzymes:*** as the myocardial necrosis occurs, the myocardial damaged cells releases cardiac enzymes into the circulation. CPK elevation appears 6 hrs aft- er MI. SGOT elevates 12 hrs after MI. LDH starts to elevate 24 hrs after MI. Cardiac specific troponin-T & troponin-I, are also elevated, starts to elevate during the first few hrs, both

are very specific to cardiac muscles, these proteins normally undetectable in the blood of healthy individuals, but rise ›20 X in pt é AMI.

③ ***Cardiac Imaging:*** ECHO: ⇩ myocardial function (⇩ EF) & significant wall motion abnormality may be detected. Radionuclide imaging.

Complications of MI

Depending on the extent of the area involved in MI, a number er of complications might arise, including the following:-

- ***Arrhythmias:*** is common on as result of hypoxia, acidosis & altered electrical conduction through damaged & necrotic areas of myocardium, may be life-threat- ening & lead to fibrillation. Lethal ventricular arrhythmia are the commonest cause of death in the first hour. This include; ventricular tachycardia or ventricular fibrillation. Atrial arrhythmias also may be seen as AF or atrial flutter.
- ***Acute conduction system abnormality:*** the conduction system may be part of the myocardium affected during infarction. This may lead to bradycardia & heart block. Inferior wall MI occurs when the right coronary artery is occluded. Since it supplies the AV node, sinus bradycardia & varying degrees of AV block may occur. In case of Anterior MI right or left bundle branch block may occur.
- ***Pump failure:*** CHF is most likely, when 30% of the myocardium is infarcted. Cardiogenic shock is defined as SBP ‹90 mmHg: occurs if ›40 % of the myocardium is affected by infarction. Cardiogenic shock is associated é a mortality rate > 80%.
- ***Mitral regurgitation:*** may occur if papillary muscles are affected.
- ***Ventricular septal defect:*** LV septum may become infracted either in anterior or inferior AMI, leading to rapture of the septum.
- ***Rupture of weakened myocardial wall:*** bleeding into pericardium may cause cardiac ta-

mponade & further impairment of cardiac pumping function. Most likely to occur é Transmural MI (involvement of full thickness of ventricular wall). Rupture of the septum between the ventricles might also occur if the septal wall is involved

- ***Left ventricular aneurysm:*** the infracted myocardium may evaginate & heal é fibrous tissue. May be a source for cardiac emboli.
- ***Pericarditis:*** post AMI pericarditis (Dressler's sy.), is believed to be autoimmune in origin. Often occurs 1-2 days after the MI.
- ***Formation of thromboembolism:*** from blood pooling in ventricles.
- ***Pulmonary infarction:*** is common on 2^{nd}-3^{rd} wk after infarction, pain, fever, haemoptysis, heart failure & pleural rub.

Management of MI

A main goal of intervention for MI is to limit the size of infarcted area & thus preserve cardiac function. Early recognition & intervention in a MI have been shown to significantly improve the outcome & reduce mortality in pts if employed in the early stages of MI. Antiplatelet-aggregating drugs such as Aspirin & clot dissolving agents such as streptokinase & tissue plasminogen activator may be very effective at improving myocardial blood flow & limiting damage to the heart muscle. Other drugs such as vasodilators, β-adrenergic blockers & ACEIs can also improve blood flow & reduce workload on the injured myocardium & thus reduce the extent of myocardial damage. The development of potentially life-threatening arrhythmias is also common during MI as consequence of hypoxia, acidosis & enhanced autonomic activity & must be treated é appropriate antiarrhythic drugs & immediate referral to hospitals é ICU facility.Management is outlined as follows:-

- ***Emegrency management:*** should start before pt reaches the hospital emergency room. A main goal of intervention is to limit the size of infarcted area & thus preserve cardiac function. Early recognition & intervention in MI have been shown to significantly improve the outcome & reduce mortality in pts. Oxygen used to maintain blood oxygenation as well as tissue & cardiac oxygen.
- ***General measures:*** reassure & make the pt comfortable. Supply O_2 by mask. Secure IV line. Give Aspirin 160-325 mg tab, if administered when MI is detected, the antiplatelet properties of aspirin may ⇩ the overall size of infarction & prevent further aggregation.
- ***Treat pain:*** Nitroglycerin: sublingual up to 3 doses of 0.4 mg should administered at about 5 min interval. Morphine sulphate: very effective analgesic for pain associated é AMI, administered in small dose of 2-4 mg IV/5 min.
- ***Limitation of infarct size through reperfusion/revascularization:*** thrombolytic agents: as Streptokinase, t-Plasminogen activator, Urokinase: these drugs are given to dissolve the occlusive thrombus & promote reperfusion of the infarct related artery, reduces mortality from MI when administered within 6 hrs of the onset of chest pain, but contraindicated é history of cerebrovascular Hge or marked hypertension or bleeding disorder. The direct per-cutaneous transluminal coronary angioplasty is the preferred method to restore perfusion of occluded coronary artery. The short & long term outcomes are much better than what can be archived through thrombolysis/or fibrinolysis.
- ***Hospital phase management:*** include, General & Specific measures;

General measures

***Activity:** absolute bed rest for the first 12 hrs, sitting on their bed in the 1st 24 hrs, the pt may ambulate in their rooms by the 2nd or 3rd day.

***Diet:** because of the risk of emesis & aspiration soon after MI, pt should receive either

nothing or only clear liquids PO for the first 4-12 hrs. Diet should be low in fat & calories & rich in potassium.

***Bowel motion:** constipation is common & straining may ppt AMI. Fibrous diet & stool softeners like Bisacodyl, Dioctylsodium Sulfosuccinate 200 mg/day are recommended. If pt. remains constipated laxatives can be prescribed.

***Sedation:** many pts require sedation to withstand the period of enforced inactivity é tranquillity. Diazepam 15-30 mg or Lorazeam 0.5-2 mg given 3-4 X daily.

Pharmacological therapy

1) ***Antithombotic & Anti platelet agents:*** unfractionated heparin: may be given in pts where there is a risk of cardiac thrombus formation &subsequent emboli. **Heparin** prev-ent clotting of blood through its action on ant thrombin 3, also inhibit formation of stable fibrin clot & has antilipaemic effect, starting dose 5000-10000 u IV followed by 1000 U/hr as continuous infusion using syringe pump é monitori-ng PT, APT. The IN to be kept 1.5-2 times of the normal value. or ***Calexane*** 40, 60, 80 u, SC/12 hrs. ***Calciparine*** 12.500 u, SC/12 hrs. ***Fraxiparine*** 0.6 ml, SC/12 hrs.

Fibrinolytic: are contraindicated for pt above 75 yrs, or pt é peptic ulcer, or bleeding disorder or major surgery in the last 2 wks, can be used é heparin during the 1st 24 hrs. Major side effects are; internal/GIT Hge, stroke & allergic reactions. Streptokinase derived from β-hemolytic streptococcus bacteria; involved in the activation of plasmin. **Kabikinase/Sidonase** during the 1st 6 hrs, 1.5million u IV infusion pump over 1hr.

Anistreplase complex of human lysplasminogen & streptokinase; administered as a prodrug. ***Alteplase*** "Rt-PA" 10 mg stat followed by 40 mg over one hour.

Urokinase endogenous human enzy that converts plasminogen to active plasmin.

2) ***Vasodilator:*** Nitroglycerin IV, ⇧blood flow to myocardium &⇩myocardial work.

Isordil/Dinitra/Angesid 10 mg tab sublingual. **Isordi**l 10 mg tab 1X1 (long acting).

Tridil: 50 mg/10 ml amp (glycerine trinitrate) IV diluted é G 5%, 1 ug/Kg/min. é careful monitoring for ⇩ of BP.

3) ***β-Blockers:*** have short & long term benefits for pts:-short term benefit as; relive pain & ⇩ the risk of arrhythmia. Long term benefits as; improving myocardial performance & facilitate healing process in post MI pts. ***Metoprolol****:* 25-200 mg BID. ***Atenolol****:* 50-150 mg PO daily. Contraindicated é severe CHF, AV block.

4) ***ACEIs:***⇩ mortality rate & improve long-term survival in post AMI by preventing cardiac remodelling ẃ may have led to progressive HF. Their effect is additive to what is archived é aspirin & β-blockers. The maximum benefit is seen in high-risk pts (elderly pt, significant LV dysfunction). ***Captopril:*** start é smaller dose 12.5mg PO/D to gradually escalate to 75mg PO BID. ***Enalapril:*** start é 12.5 mg PO daily, escalate gradually to 40 mg PO/day.

5) ***Aspirin/Plavix:*** inhibits the cyclooxygenase pathway for the synthesis of prostaglandi-ns, prostacyclins & thromboxanes. Inhibits aggregation of platelets & is effective in redu-cing MI, stroke & mortality in high-risk pts.

Aggrex 75 mg tab. 1 X 1 daily, or ***Plavix*** tab. 1X1 daily.

6) ***Marevan*** after discharge from hospital, maintenance therapy 1 mg tab daily, follow up by INR to keep INR from 2-3.

7) Ca.Ch.Bls, ***Delaytiazim*** 90, 120 mg tablet 1 X 1.

8) ***Antioxidant: Omega 3*** 1 X 1 daily.

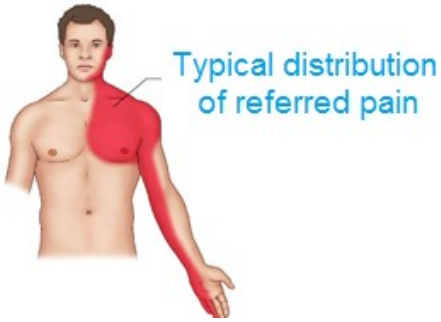
9)***Lipid regulating agent, Zocor*** tab. 1 X 1 daily to keep cholesterol ˂ 250 & triglyceride ˂200 & LDL ˂100, & HDL ˃35. Lipid profile to be repeated monthly.

Management of complications

- ***Malignant arrhythmias:*** cardiac defibrillation. Prophylactic lidocaine. Other antiarrhythmic agent as; Bertyluim, Tosylate & Procainamide.
- ***Serious conduction disturbances:*** sinus bradycardia: Atropine 2 mg IV may restore heart rate. AV block: transcutaneous pace makers.
- ***Heart failure:*** diuretics, salt restriction, ACEIs, vasodilators. The use of Digoxin is controversial.
- ***Cardiogenic shock:*** do ECHO to assess the ventricular function. IVF to maximize LV filling. Use of vasopressors (Dubutamine, Dopamine) in IV infusion. Intra-aortic balloon pumping. Percutaneous transluminal angioplasty.

Prognosis

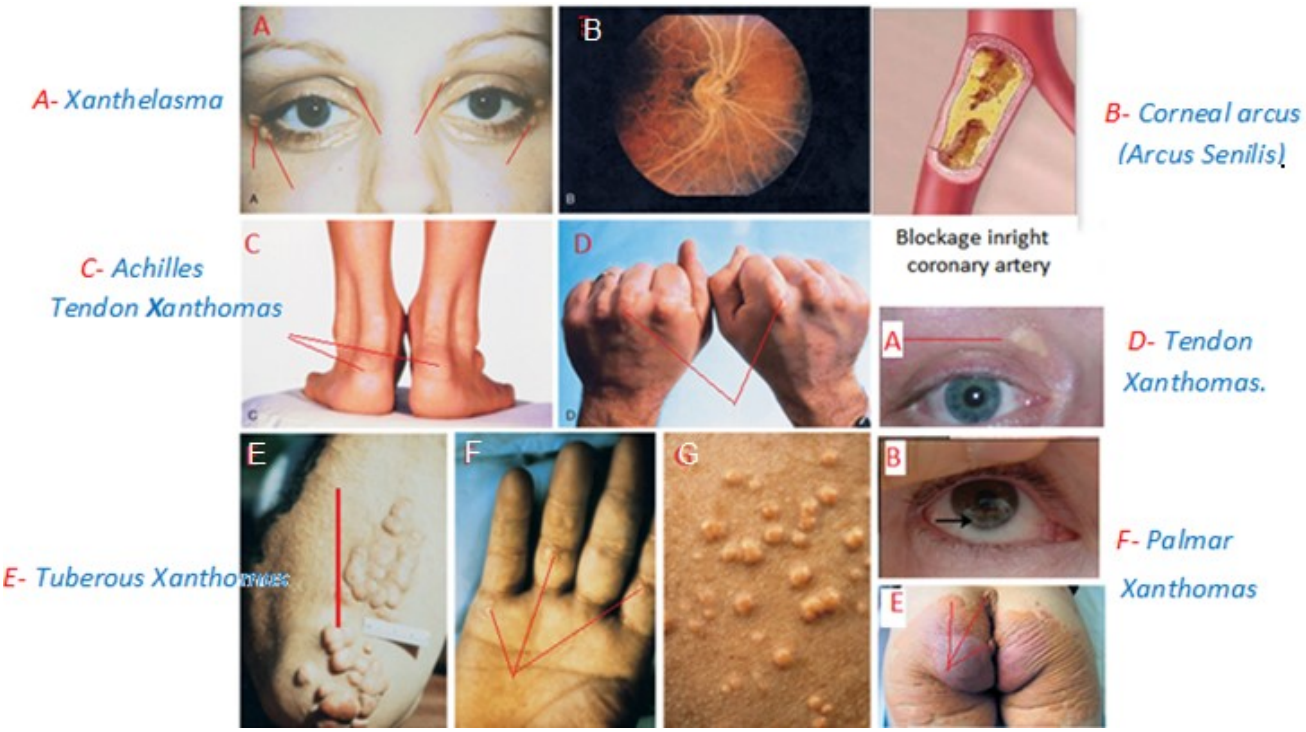
Depends upon number of vessels affected & extent of ventricular damage. Pt é uncorrected main left coronary artery disease have 20% mortality in the 1st year. Single vessel coronary artery has 2% annual mortality. Double vessel disease has 2-4% annual mortality. Triple vessel disease has 5-8% annual mortality. The left ventricular EF of **<** 40% doubles the yearly mortality.



FAMILIAL HYPERCHOLESTREMIA

AD disorder, characterized by high levels of LDL & early coronary artery disease. Heterozygous ~1 in 500 & Homozygous ~1/1,000,000. Greater risk of heart disease (100 X for males 20-40 yrs). 85% of affected individuals remain undiagnosed.

Diagnosis: in addition to ⇧ cholesterol, the following manifestations may be seen:



Suspicions & Screening:

Who to screen?

▲Anyone é high cholesterol by age 20. ▲All children aged 9-11 ▲Children as young as 2 yrs é family history of premature cardiovascular disease or very high cholesterol levels.

Who to suspect?

■Adults aged 20 or older é LDL cholesterol >190 mg/dl.

■Children aged 9-11 yrs é LDL cholesterol 160 mg/dl.

Management

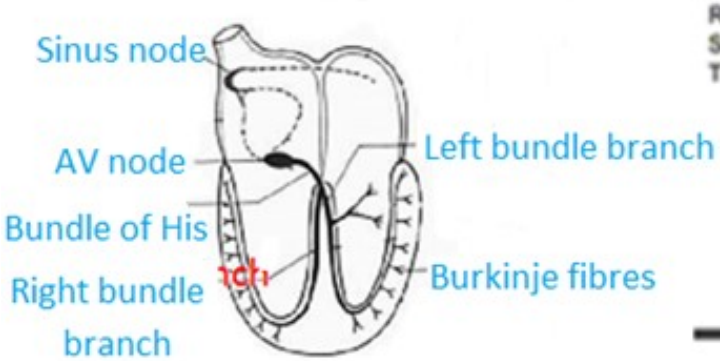
•Early identification •Reduction in morbidity through changes in the lifestyle. Diet, exercise & no smoking • Use of statins to lower cholesterol.

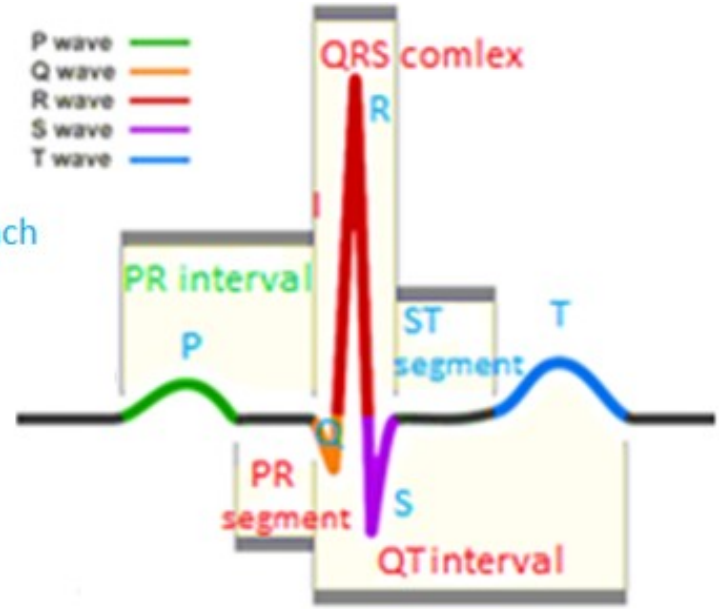
CARDIAC ARRHYTHMIAS

Cardiac arrhythmias are changes in the regular beating of the heart. The heart may seem to skip a beat or beat irregularly or beat very fast or very slow. Normal heart beat of a person ranges from 60-100/min. Many arrhythmias occur in people who do not have underlying heart disease. Most of the time, there may not be a recognizable cause of an arrhythmia. Heart disease may cause arrhythmias. Other causes include; stress, caffeine, tobacco, alcohol, cough & cold medicines. In a very small number of people é serious symptoms, arrhythmias themselves are dangerous. These arrhythmias require medical Rx to keep the heart beat regular. Arrhythmias occur commonly in middle age adults. As people get older, they are more likely to experience an arrhythmia. Pts é arrhythmias often complain that they felt their heart beat very fast, experienced a fluttering in their chest, or noticed that their heart skipped a beat. Almost everyone has also felt dizzy, faint, or out of breath or had chest pain at one time or another. Also arrhythmia seen in 40% of premature babies.

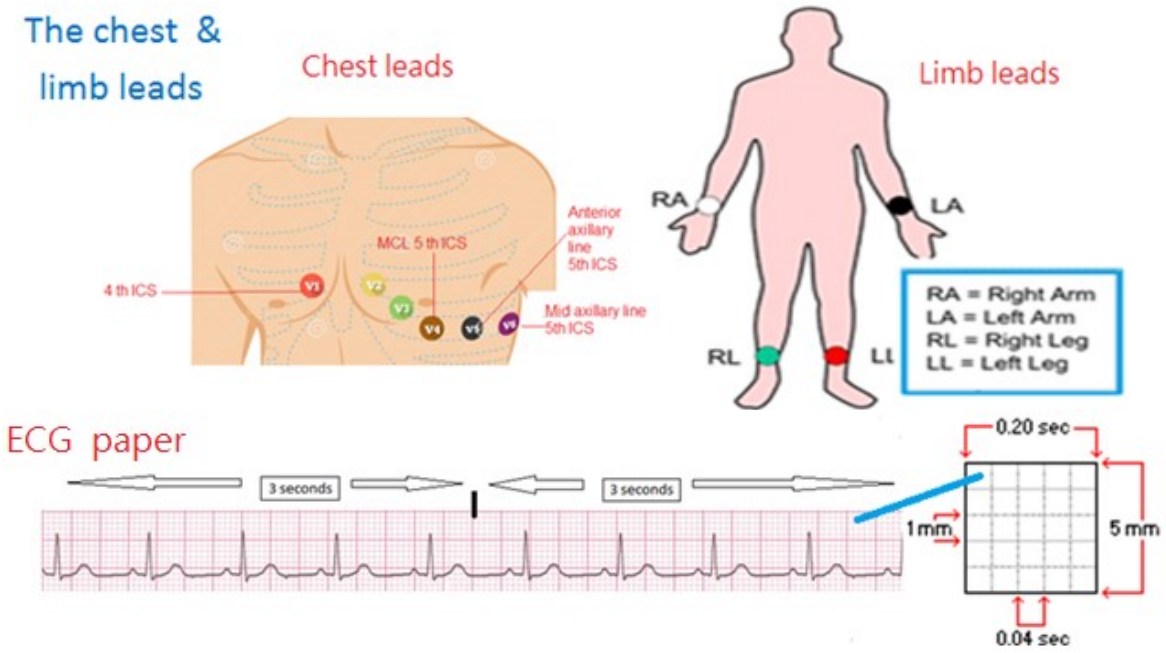
Normal impulse conduction

SAN ⇨ AVN ⇨ Bundle His ⇨ Bundle branch ⇨ Purkinje Fibres





Doing an ECG



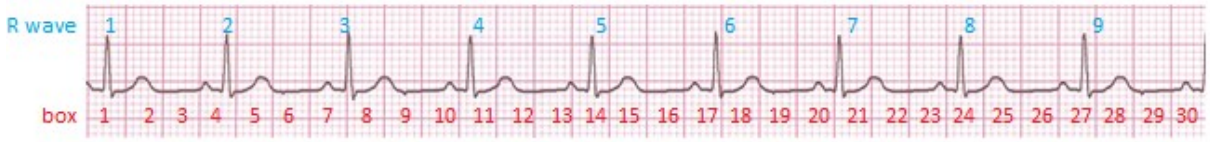
Practical point to remember on interpretation of an ECG:-

- P wave: represent atrial depolarization.
- QRS: represent ventricular depolarization.
- T wave: re-present ventricular repolarization.
- Small box = 0.04 sec. (1 mm).
- Large box = 0.2 sec. (5 mm or ½ mv).
- Every 15 large boxes in the strip =3 sec. This helps when calculating the HR.
- P wave: normally < 2 small squares & its peak is < 2 small squares. A Wider bifid P wave means LAH. A peak of P wave> 2 small square means RAH.
- P-R interval: normally < 4 small squares (4 mm).
- R wave: normally 2 mm height.
- QRS wave: normally 3 mm width, become broader in Heart Block.
- S-T segment elevation: seen in MI, Pericarditis, or Ventricular Aneurysm.
- S-T segment depression: seen in Hypertension, IHD, LVH, Hypokalaemia, Hypocalcemia emia, or Digitalis toxicity

How to read an ECG

Step 1: Calculate Rate

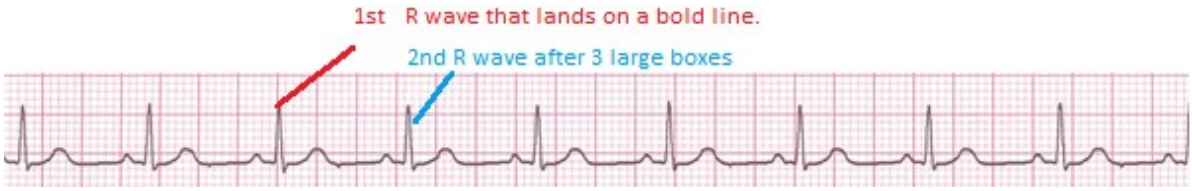
Option 1:



Count number of R waves in 6 second rhythm strip, (30 large boxes) then multiply by 10.

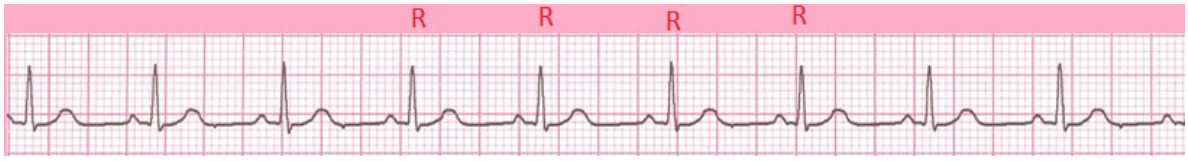
Interpretation of the above strip rate *9 x10 = 90 bpm.*

Option 2:



Find a R wave that lands on a bold line. Count number of large boxes to the next R wave. If the 2nd R wave is 1 large box away the rate is 300, 2 boxes away the rate is 150, 3 boxes -100, 4 boxes -75, etc.

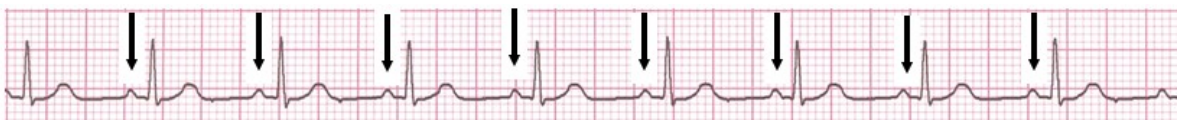
Step 2: Determine regularity



Look at the R-R distances (using markings on a pen or paper). Is it regular ? or occasionally irregular? or regular irregularity ? or it is irregular irregularity?

Interpretation of the above strip: regular rhythm.

Step 3: Assess the P waves

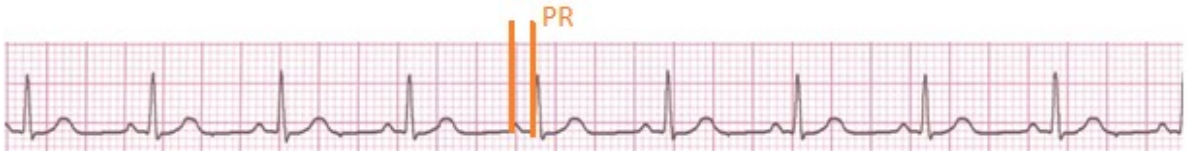


Are there P waves? Do the P waves all look alike? Do the P waves occur at a regular rate?

Is there one P wave before each QRS?

Interpretation: the above ECG shows normal P waves é one P wave for every QRS.

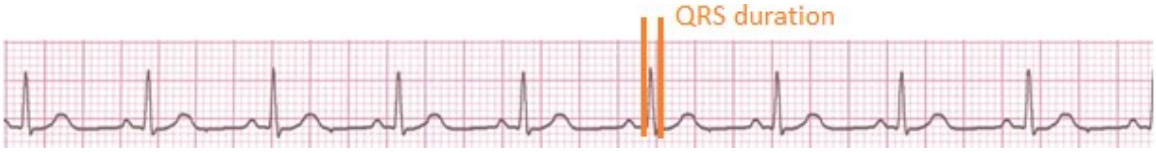
Step 4: Determine PR interval



Normal PR interval = 0.12-0.2 seconds (3-5 small boxes = 3-5 mm).

Interpretation: normal PR interval (0.12 seconds).

Step 5: Determine QRS duration



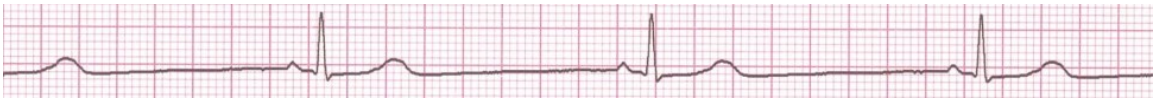
Normal QRS= 0.04-0.12 seconds (1-3 small boxes = 1-3 mm).

Interpretation: normal QRS (0.08 seconds).

RECOGNIZE THE 13 MOST COMMON RHYTHM DISTURBANCES.

Look to each of the following strips & try to interpretate.

1-Sinus Bradycardia



Rate: 30 bpm. -Regularity: regular. -P waves: normal. -PR interval: 0.12 second. - QRS duration: 0.10 second.

Interpretation: *Sinus bradycardia*

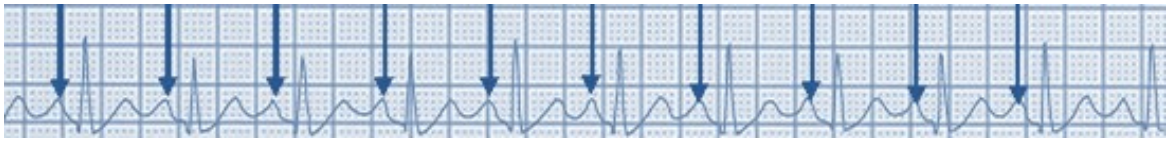
Comment: deviation from normal sinus rhythm, the HR is ˂60 bpm, SA node is depolarizing slower than normal, impulse conducted normally (i.e. normal PR & QRS interval).

Causes: •Physical conditioning in professional athletes. •Hypothyroidism. •Sinus node dysfunction.

Therapy:

- If it is physiologic no need for treatment.
- If it is due to sinus dysfunction & severe (the HR is < 35 bpm):-
  - Atropine: 1 mg IV may temporarily ⇧ the sinus rate.
  - Cardiac pacemaker implantation.

2- Sinus Tachycardia



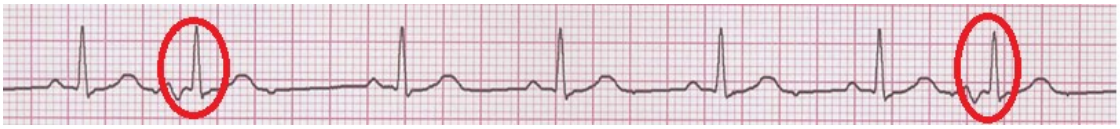
Rate: 130 bpm. -Regularity: regular. -P waves: normal. -PR interval: 0.16 sec.-QRS duration: 0.08 second.

Interpretation: Sinus tachycardia

Comment: the SA node depolarizing faster than normal, impulse is conducted normally (SA node sends out electrical signals faster than usual, speeding HR).

Causes: it represents physiologic sinus tachycardia in response to physical or psychologic-al stress. Rate rarely exceeds 200 bpm.

3-Premature Atrial Contractions



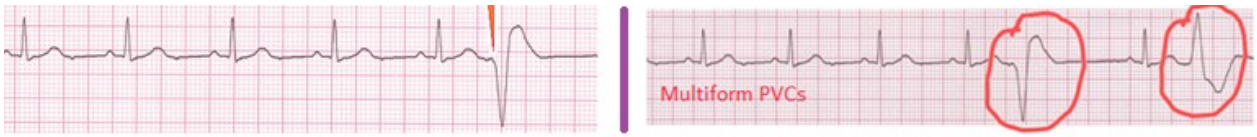
Rate: 70 bpm -Regularity: occasionally irregular -P waves: beat 2 & 7 have different cont-our -PR interval: 0.14 second (except beat 2 & 7) -QRS duration: 0.08 second.

Interpretation: Normal sinus rhythm é PACs.

Comment: these ectopic beats originate in the atria (but not from SA node), therefore the contour of P wave, PR interval & the timing are different than a normally generated pulse from SA node. The excitation of an atrial cell forms an impulse that is then conducted

normally through the AV node & ventricles.

4- Premature Ventricular Contractions



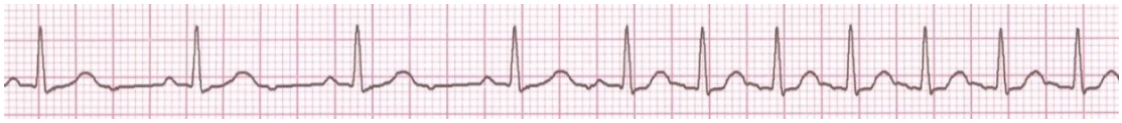
Rate: 60 bpm. -Regularity: occasionally irregular. -P waves: 6 are normal & no P wave for the 7th QRS. -PR interval: 0.14 sec. QRS: duration is 0.08 sec. & the 7th QRS is wide.

Interpretation: *Sinus Rhythm é one PVC. (in left strip & multiform in the right)*

Comment: ectopic beats originate in the ventricles resulting in wide & bizarre QRS complexes. When there are ›1 PVCs & look alike, they called "uniform". When they differ they called "multiform". One or more ventricular cells are depolarizing & the impulses are abnormally conducting through the ventricles. When an impulse originates in a ventricle, conduction through the ventricles will be inefficient & the QRS will be wide & bizarre. PVCs are among the commonest arrhythmias.

Therapy: most of the isolated PVCs are benign & need no treatment.

5- Paroxysmal Supraventricular Tachycardia



Rate: variable, 74 ⇨ 148 bpm - Regularity: regular ⇨ irregular. - P waves: normal ⇨ none. - PR interval: 0.16 sec. ⇨ none. - QRS duration: 0.08 sec.

Interpretation: Paroxysmal Supraventricular Tachycardia

Comment: series of early beats in the atria speed up the heart rate. Heart rate of 150-250 bpm, occur as repeated periods, begin & end suddenly. Often occurring in pt é otherwise normal heart. The incidence ⇧ é age & é the presence of cardiovascular diseases. The Incidence of new cases is 35/100.000 persons/year.

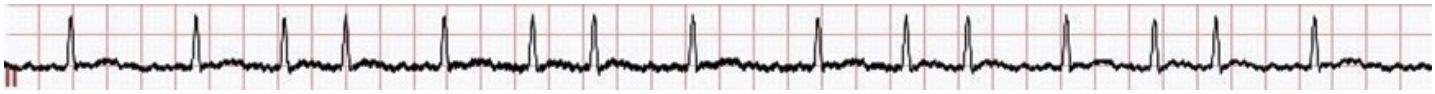
Causes: •Exercise •Fever •Anxiety •Thyrotoxicosis •Hypoxemia or •Hypotension.

Clinical picture:Palpitations: trigger is usually not identified. Feeling of heart pounding in chest & neck. Anxiety, light headedness, dyspnea. Syncope & chest pain are uncommon, but may indicate coronary artery disease, especially in older pts. Psychological stress is very common.

Diagnosis: •ECG. •Holter 24 hrs monitor.

Treatment: •***If pt is stable:*** Identify & treat the underlying cause. •***If pt hemodynamically unstable:*** Mechanical Rx; carotid massage, to one side of neck because massage to both sides may cuse cerebral stroke, valsalva manoeuvre, head immersion in cold water. Medical Rx; β-blockers, Ca Ch BL. (Verapamil & Diltiazem). Digoxin 20 ug/Kg/ D÷3 IM effective in 60-80% of pts. For chronic SVT, class 1A or 1C or Amiodarone work well. Inderal 40 mg 1X2 also effective. Ablation will cure it too, but usually done for young pts.

6- Atrial Fibrillation



Rate:? 100 bpm. - Regularity: irregular irregularity. - P waves: none. PR interval: none - QRS duration: 0.06 second.

Interpretation: Atrial Fibrillation

Comment: electrical signals in the atria are fired in very fast, uncontrolled manner. The electrical signals arrive in the ventricles in a completely irregular fashion & heart beat is completely irregular. No organized atrial depolarization, so no normal P waves as the impulses are not originating from the SA node. The atrial activity is chaotic res-ulting in irregular irregularity rate. Recent theories suggest that it is due to multiple reenterant wavelets conducted between the Rt & Lf atria. Either way, impulses are formed in totally

unpredictable fashion. The AV node allows some of the impulses to pass through at variable intervals. 25% of people aged 40 yrs or older develop AF. Men affected > women & the number of people é AF is expected to double by year 2050. The heart beats are completely irregular, often too fast or too slow.

Causes: whilst some cases of AF have no known cause, conditions & life style factors known to lead to AF include:- •Age **>** 40 yrs. •Poorly controlled hypertension •HF. •DM. •Atherosclerosis. •Thyrotoxicosis. •Stress. •Fever. •Excessive alcohol intake. •Hypotension. •Endocarditis. •Pericarditis. •CAD. •MI. •Open heart surgery. •Pulmonary embolism. •Cold •Mitral valve diseases. •Rheumatic heart. •Sick sinus syndrome •Obesity. •Iatrogenic or •Idiopathic.

Symptoms

May be experienced on regular basis, intermittently or not at all. **>**50**%** of episodes of AF are not felt by the pt. Symptoms include; fatigue, palpitations, dizziness, chest pain & breathlessness. Asymptomatic AF is substantial problem for individual health & the health care system: it may cause stroke. it is frequent despite antiarrhythmic drug Rx or catheter or surgical ablation. It may cause cognitive dysfunction & dementia.

Complications

- Stroke.
- Mesenteric ischemia.
- Claudications of lower limbs.

How does AF lead to stroke?

Blood pools in the atria ⇨ Blood clot forms ⇨ Whole or part of the blood clot breaks off ⇨ Blood clot travel to the brain & closes a cerebral artery ⇨ Stroke.

What is a stroke?

Stroke is the brain equivalent of MI. Blood must flow to & thro-ugh the brain for the brain to work properly. If this flow blocked by clot, brain los-es its energy &O_2 supply, causing brain damage that can lead to disability or death. People é AF are 5 times more likely to have stroke, while 20-30% of strokes are related to AF.

Classification of AF

⊙ ***Paroxysmal AF***: terminates in ˂7 days.

⊙***Persistent AF***: fails to terminate within 7 days.

⊙***Permanent AF***: ˃1 yr.

⊙***Lone AF***: individuals éout structural heart disease, & ˂60 yrs.

EHRA score for AF (according to symptoms)

	Explanation
EHRA I	No symptoms
EHRA II	Mild symptoms, normal daily activity not affected
EHRA III	Severe symptoms, normal daily activity affected
EHRA IV	Disabling symptoms, normal daily activity discontinued

Diagnosis

HR 100-175/min, irregular, can`t count 4 consequent regular heart beats. Pulse deficit ≥ 10 beats difference between the radial pulse & auscultation. Difference in intensity of the 1st HS, absence of A wave in JVP & the ECG changes.

Management

The goal is to achieve rest HR 60-80 bpm & activity HR 80-110 bpm.

If pt hemodynamically unstable: direct current synchronous cardioversion: 2 watt /Kg/second, may repeated 2-3 times é duplication of dose.

If pt hemodynamically stable: identify & treat the underlying cause. Control the ventricular rate: β-blockers, Ca Ch BL, Digoxin. Restore sinus rhythm: Quinidine.

Antithrombotic therapy: anticoagulant & Antiplatelet medications. The Anticoagulant should be recommended in every pt é persistent or paroxysmal AF unless clinically contr-aindicated. The INR control goal is to keep it at a range of 2-3.

Reasons for not prescribing anticoagulation

•Advanced Age. •High falls risk. •Dementia. •Poor INR control. •Atrial Flutter. •Frailty. •History of previous significant Hge or risk factors for bleeding.

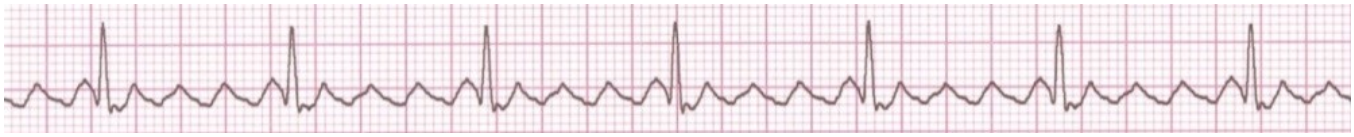
Antiplatelet: near-complete platelet inhibition is achieved ē Aspirin 75 mg daily.

Major issues at present

- Early Rx by rhythm control therapy?
- Anti-arrhythmic drugs Vs catheter ablation?
- Better prevention by novel drugs, health care costs.

Category	Drug	IV dose	Oral (maintenance)
B Blockers	Metoprolol CR/XL	2.5-5 mg bolus over 2 min up to 3 doses	100-200 mg
	Bisoprolol		2.5-10 mg
	Atenolol		25-100 mg
	Esmolol	50-200 ug/kg/min	
	Propranolol	0.15 mg/kg over 1 min	10-40 mg tid
	Carvedilol		3.125-25 mg bid
Ca Ch Bls	Verapamil	0.0375-0.15 mg/kg over 2 min	40-360 mg bid
	Diltiazem		60 -360 mg tid
Digitalis	Digoxin	0.5- 1 mg	0,125 mg- 0.5 mg
	Digetoxin	0.4 – 0.6 mg	0.05 mg-0.1 mg
Others	Amiodarone	5 mg/kg in 1 hour	100mg-200 mg

7-Atrial Flutter



Rate: 70bpm -Regularity: regular -P waves: flutter waves. -PR interval: none. -QRS 0.06sec

Interpretation: Atrial Flutter

Comment: no P waves, instead flutter waves (note saw tooth pattern) are formed at a rate of 250-350 bpm. Only some impulses conducted through the AV node (strip: every 4 P waves one QRS regular.

Etiology: reentrant pathway in right atrium é every 2^{nd}, 3^{rd} or 4^{th} impulse generating a QRS, others are blocked in the AV node as the node repolarizes. Sometimes there may be a varying block resulting in irregular ventricular beat.

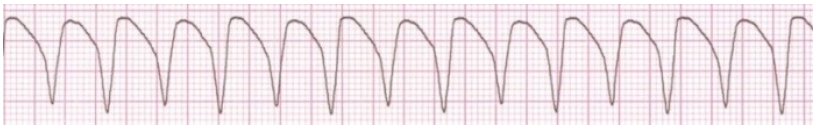
Causes: often associated é antecedent heart diseases:-

•Coronary artery diseases. •Pericarditis. •Valvular heart diseases. •Cardiomyopathy.

Therapy: •***Direct current cardioversion:*** if pt is hemodynamically unstable.

•***Drugs:*** Digoxin, Esmolol or Verapamil. Quinidine to restore sinus rhythm.

8. Ventricular Tachycardia



Rate: 160 bpm. -Regularity: regular. -P waves: none. -PR interval: none. -QRS dur-ation: wide (**>** 0.12 sec).

Interpretation: Ventricular Tachycardia

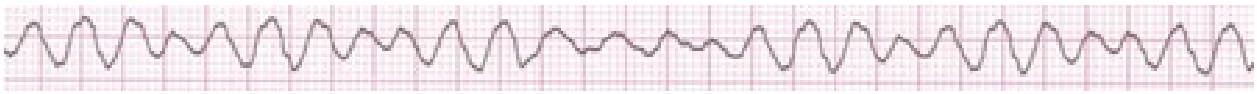
Etiology: there is a reentrant pathway looping in a ventricle (the most common cau-se). It occurs paroxysmal & exceeds 120 bpm é regular rhythm. There is AV dissociation & the ventricular arrhythmia proceeds independently of the normal atrial rhythm. During ventricular tachycardia, the ventricles do not have enough time to relax, ventricular filling is impaired & COP significantly ⇩. Ventricular tachycardia can sometimes generate enough COP to produce pulse; at other times no pulse can be felt. When ventricular tachycardia lasts for ›30 sec or requires control because of hemodynamic collapse it is

called sustained ventricular tachycardia. Ventricular tachycardia may quickly degenerate to ventricular fibrillation & death.

Therapy: since this is a life threatening situation, urgent intervention needed.

Anti-arrhythmic drugs: IV Beretylium, Lidocaine or Procainamide may be useful in returning the pt's rhythm to normal while preparation is being made for ***DC cardiove-rsion*** ẃ urgently required.

9. Ventricular Fibrillation



Rate: none. -Regularity: irregular irregularity. -P waves: none. -PR interval: none.

- QRS duration: wide, if recognizable.

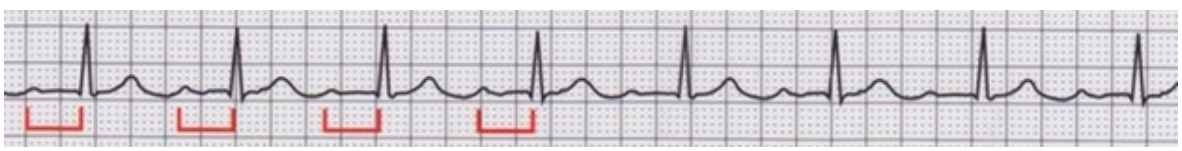
Interpretation Ventricular fibrillation

Etiology: electrical signals in the ventricles are fired in a very fast uncontrolled mann-er, causing the heart to quiver rather than beat. It is characterized by lack of ordered contraction of the ventricles. Therefore there is no COP, thus ventricular fibrillation synonymous é death unless urgent conversion to effective rhythm can be accomplished.

Therapy

- Cardiac resuscitation.
- Mechanical ventilation.
- Intracardiac adrenalin.
- DC cardioversion using high voltage.

10- First Degree AV Block



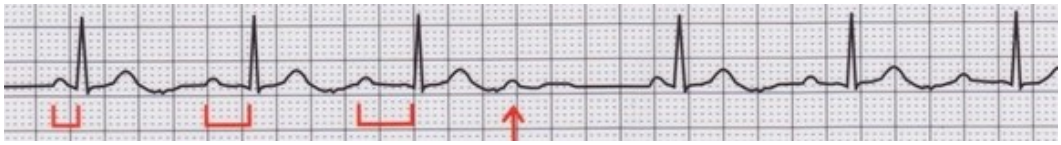
Rate: 60 bpm. -Regularity: regular. -P waves: normal. -PR interval: 0.36 sec. -QRS duration: 0.08 second.

Interpretation: First degree AV Block

Etiology: prolongation of PR interval > 0.2 second.

Therapy: we use Dopamine, or Isuprel.

11- Second Degree AV Block, Mobitz Type I (Wenckebach)

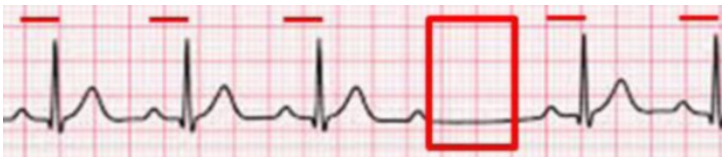


Rate: 50 bpm - Regularity: regular-irregularity - P waves: normal, but no QRS on the 4th beat - PR interval: progressively lengthens - QRS duration: 0.08 sec.

Interpretation: Second degree AV Block, mobitz type I.

Comment: PR interval progressively lengthens,then the impulse is completely blocked (P wave not followed by QRS).

12. Second Degree AV Block, Mobitz Type II

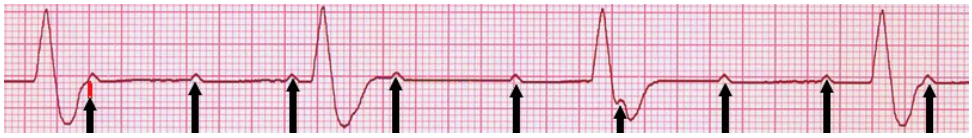


Rate: 40 bpm -Regularity: regular -P waves: normal & on the 4th beat no QRS - PR interval: 0.14 sec. - QRS duration: 0.08 sec.

Interpretation: 2nd degree AV Block Type II

Comment: occasional P waves are completely blocked (the P wave not followed by QRS). - Intermittent drop of QRS, can rapidly progress to complete heart block. No prolongation of PR interval before the dropped beat. Typically block occurs in the bundle of His.

13. Third Degree AV Block



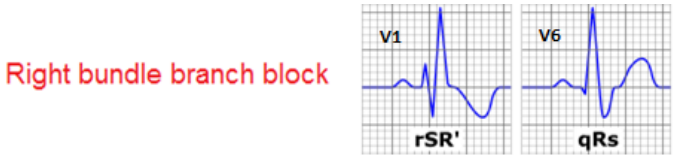
Rate: 40 bpm. -Regularity: regular. -P waves: no relation to QRS. PR interval: none. -QRS: wide (**>** 0.12 sec). -The atrial rate **>** 100 bpm & ventricular rate is 38 bpm.

Interpretation: 3rd Degree AV Block

Comment: no atrial impulses conducted. Both atria & ventricles are contracting independen- tly. The ventricles own intrinsic pace maker kicks in at around 30-45 bpm & pts become symptomatic. No relation between P & QRS.

Therapy: pharmacologic therapy reserved only for acute situations for temporarily ⇧ of the ventricular rate. Atropine 0.5-2 mg IV or Isoproterenol 1-4 microgram/ min IV. Permanent cardiac pacemakers for most symptomatic AV blocks.

Right Bundle Branch Block

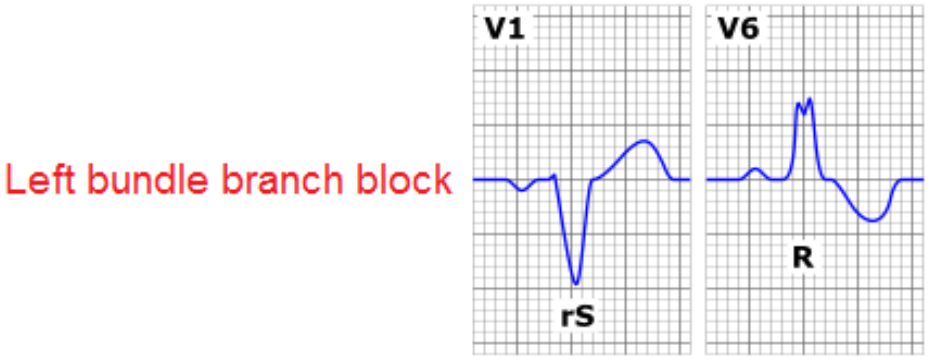


How is the right ventricle depolarized in RBBB? Impulses from the left ventricle; the septum depolarizes from left to right, the left ventricle is depolarised as norm al, the right ventricle is depolarized late & in an anterior direction. The QRS is wide due to slow conduction through myocardial cells.

ECG changes in RBBB: QRS duration exceeds 0.12 sec. RSR1 complex in V1. Delayed slurred S wave in I, aVL, V5 & V6. ST/T components are opposite in direction to the terminal QRS (this is 2ry to the block & does not predispose primary ST/T).

Significance of RBBB: Normal subjects ”occasionally”. Pulmonary embolus. Corona ry artery disease. ASD. Active cardities. Right ventricular diastolic overload.

Left Bundle Branch Block



How is the right ventricle depolarized in RBBB? the left ventricle is activated from the RBB & right ventricle. From the above diagram;

1a. Impulses pass to the left of the septum, therefore depolarizing it from Rt to Lf.

1b. Impulses travelling down the RBB simultaneously depolarize paraseptal region.

2. RV depolarization follows, small magnitude.

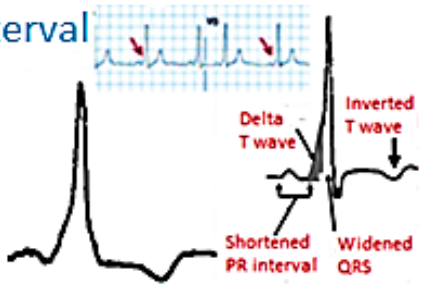
3. Delayed LV depolarization due to slow conduction through myocardium.

ECG changes in LBBB: QRS duration exceeds 0.12 sec. Wide, notched (M shaped) QRS in I, aVL, V5 & V6 (LBBB is best seen in V6). Wide, notched QS complexes in V1. Small r in V2 & V3 - paraseptal depolarization.

Occurs in? always indicative of organic heart disease. In IHD. In hypertension.

Wolf Parkinson White Syndrome

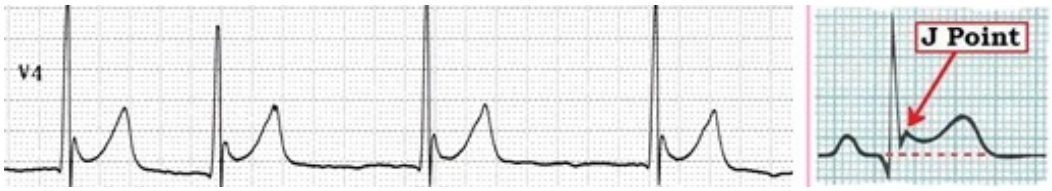
ECG shows: delta wave, short P-R interval widened QRS complex.



WPW is an abnormal band of atrial tissue connects the atria & ventricles & can electrically bypass the normal pathways of conduction. A reentry circuit can develop causing paroxysms of tachycardia.

Management: drugs includes Flecainamide, Amiodarone or Disopyramide. Digoxin & Verapamil are contraindicated. Transvenous catheter radiofrequency ablation (is the treatment of choice).

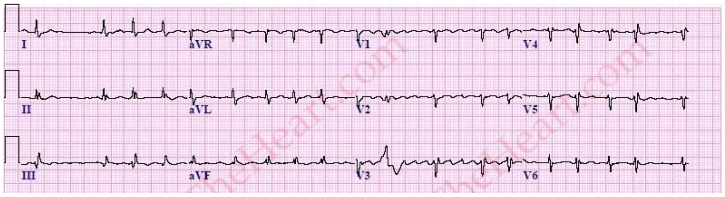
J Wave



ECG shows hump like wave superimposed on QRS distal limb (J Point). Bradycardia & Osborn waves (J-waves) all over.

Causes: •Hypothermia (temp < 30 ^{0}C), but not pathognomonic, as J waves may be seen as a normal variant. •Hypercalcaemia. •Medication. •Head injury. •SAHge.

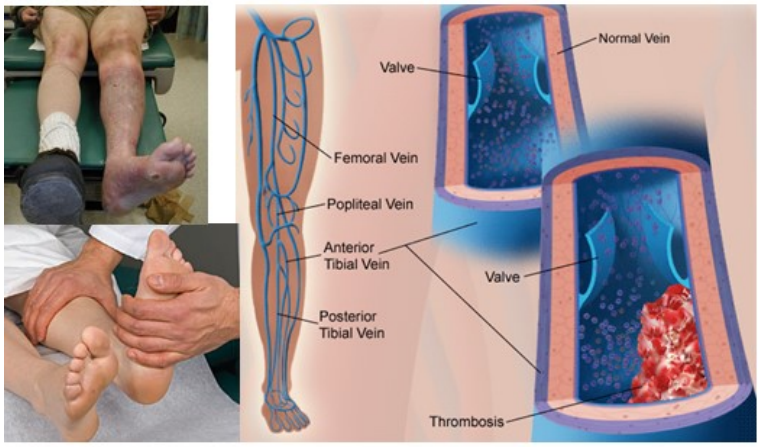
LOW VOLTAGE ECG



Seen in case of:-

- Myxedema.
- Pericardial effusion.
- Constrictive pericarditis.
- Emphysema.
- Incorrect standardization.

VENOUS THROMBOEMBOLISM



Is a spectrum include (1) Deep venous thrombosis. (2) Pulmonary embolism.

Incidence: 1/1000 (⇧ é age).

Risk factors: •Stroke •Heart disease •Hyperlipidemia •Smoking in females •Obesity •Cancer •+ve family history •Recent surgery, particularly orthopaedic, within past 4wks •Serious illness: sepsis, severe infection, ulcerative colitis •Polycythemia •Spinal cord injury, burns, lower extremity fractures •Contraceptives/oestrogen Rx & pregnancy •Paralysis or immobility for >3 days •Plane or car travel (>4 hrs).

Deep venous thrombosis in upper & lower extremities

Formation of a blood clot that does not break down, in a deep vein of the body. It can become large & obstruct the normal flow in the vein. Deep veins of the LL are the most common sites. If the clot breaks into smaller pieces, it becomes an embolus, travel to vital organs & cause heart attack, stroke, or pulmonary embolism.

Clinical picture

•Sudden swelling in the affected limb. Calf tenderness or limb pain.

•+ve Homan's sign (discomfort in calf muscles on forced foot dorsiflexion é knee straight, but Homan's sign is neither sensitive nor specific & is present only in <1/3 of pts é confirmed DVT & found in 50% of pts éout DVT.

•Dilated superficial collateral veins, minute petechiae/ecchymosis.

•Cyanosis or pallor of the leg.

•Leg may be cool é diminished arterial pulsation (distal pulse of dorsalis paedis, posterior saphenous & anterior tabialis).

•The skin over the area of thrombosis may be warm. Often difficult to differentiate from non-thrombotic disorders.

Diagnosis

•Pt risk factors/medical history physical examination.

•Specific limb symptoms (oedema, pallor). •Duplex U/S. •MRI. •Venography.

•D-dimer is a fibrin degradation product, a small protein fragment present in the blood after a blood clot is degraded by fibrinolysis. It is so named because it contains two cross linked D fragments of the fibrin protein.

Complications

•Pulmonary embolism (in 1/3 of pts) •Post-thrombotic syndrome leading to chronic venous insufficiency or ulcers & critical limb ischemia.

Management

Anticoagulation: (Heparin, Warfarin, LMWH). Reduces occurrence of a pulmonary embolism, can ⇩ symptoms. Its disadvantages include; bleeding from long-term use & it does not ⇩ the thrombus burden. ***Thrombus removal:*** catheter directed thro-mbolytic therapy. ***Compression stockings*** prevent post-thrombotic sy.

HEART & ELECTROLYTES

The electrolytes potassium, magnesium, sodium & calcium play a crucial role in the function of the myocardium, the muscular tissue of the heart. Movement of these ions across the semi-permeable myocardial cell membrane causes the voltage across the membrane to exceed a threshold & generate an action potential, resulting in muscle contraction. Electrolytes carry electrical charge & are maintained to tight physiological concentrations through various mechanisms to ensure appropriate heart function. An imbalance of these electrolytes can have detrimental effects on the heart, causing or contributing to arrhythmia & cardiac arrest. Life-threatening arrhythmias are commonly associated é potassium disorders, particularly hyperkalemia in ẃ the potassium level is elevated, less com-monly é disorders of serum calcium & magnesium. Electrolyte imbal-ances also have wider effects in the body.

Electrolytes

Work é fluids to keep the body healthy & in balance. They are solutes that are found in various concentrations & measured in terms of mille equivalent (mEq) units. Are either +ve charged (cations), as (Na^+), (K^+), (Ca^{++}), (Mg^{++}) & (H^+), or -ve charged (anions), as (Cl^-), (PO_4^-), (HCO_3^-) & (SO_4^-). For homeostasis body needs: total body anions = total body cations. The Non-electrolytes include; Glucose, Urea, Protein, Lipids etc….

Body fluids

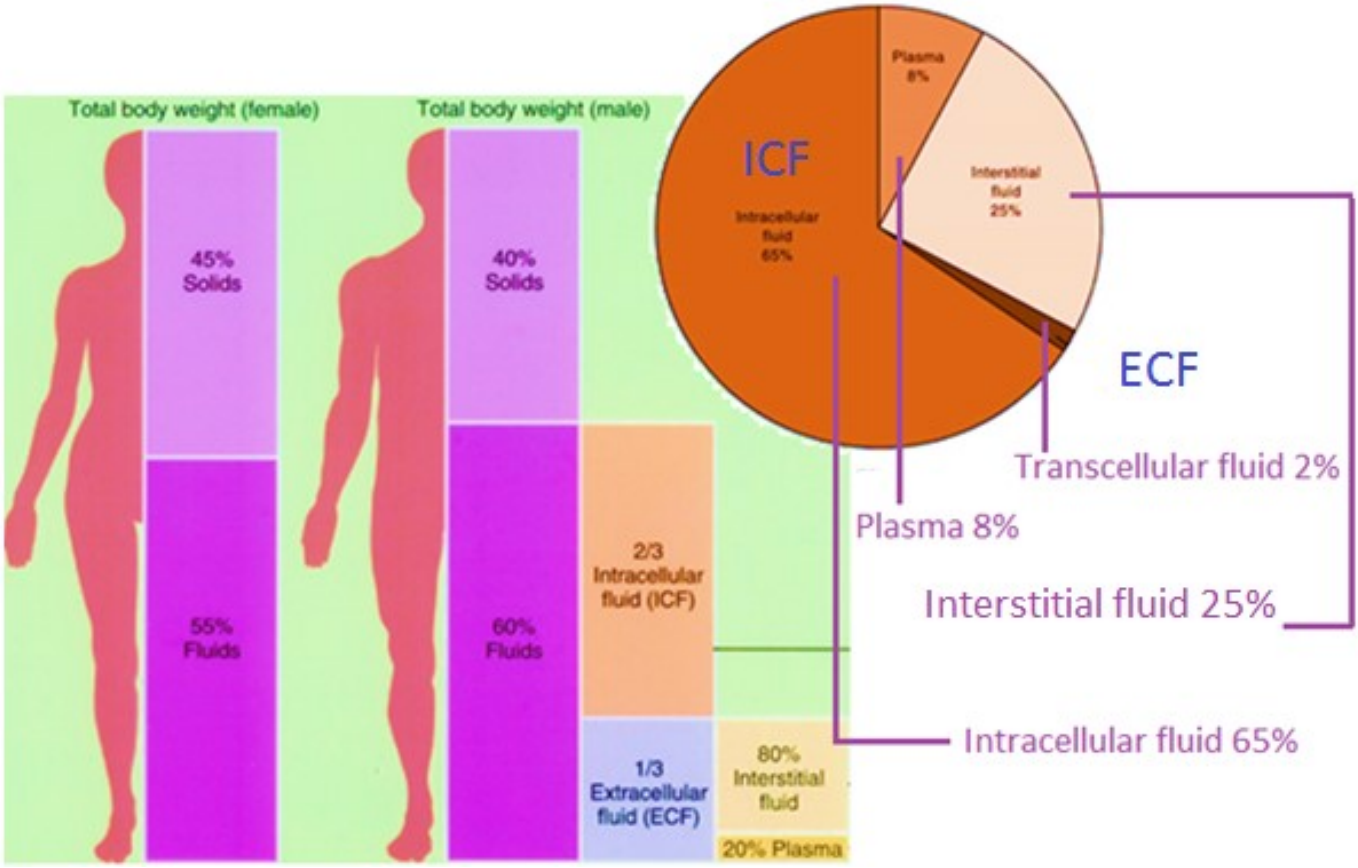
Varies é weight, age & sex; in early embryo it is (97%), in Newborn it is (77%), in Adult male it is (60%) & in Adult female (54%), while in the elderly (45%).

Function of body fluids: water is the most important nutrient for life, fluid. Act as medi-um for transport, needed for cellular metabolism, solvent for electrolytes & other constit-uents, helps to maintain body temperature, helps digestion & acts as a lubricant.

Mechanism of Gain & Loss of body fluids: the gain is through (for adult); fluid intake about 1500 ml, food intake about 1000 ml & Oxidation of nutrients 300 ml (10 ml of H_2O/100 Kcal). The losses are through the "Sensible", can be seen include; urine 1500 ml, sweat 100 ml. "Insensible", not visible, include; skin (evaporation) 500 ml, lungs 400 ml, faeces 200 ml. The loss of 10% body fluid (8% wt. loss) is serious, loss of 20% body fluid (15% wt loss) is fatal. The fluid gained each day should be equal to the fluid lost & each is equal to 2 -3 Litres/day on average.

Compartments of body fluids

- Interstitial (fluid around/between cells).
- Intravascular (plasma fluid in blood vessels).
- Transcellular (as in CSF, synovial fluid etc..).



HYPERKALAEMIA

- Hyperkalemia = plasma K^+ conc. > 5.0mmol/L
- Critical hyperkalemia = plasma K^+ conc. > 6.5 mmol/L

Causes: •Acute/chronic renal failure. •Metabolic acidosis •Hyperglycaemia. •Thrombocytosis. •Leucocytosis. •Trauma. •Burn. •Prolonged tourniquet (muscle cells relea- se K as result of tissue anoxia). •Digoxin toxicity. •Succenyl Choline (anaesthesia). •Non selective ß-blockers as Propranolol, Labetalol •ACEIs • NSAIDs & •Lab error.

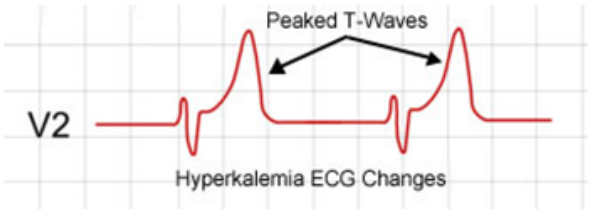
Clinical picture: •Vague •Weakness •Flaccid paralysis •Malaise •Lethargy.

ECG changes

*No P wave.

*Tall peaked T wave

* Widened QRS



Management

•Ca gluconate amp 10%, 10 ml over 2-5 min., or •Glucose 50%, 1 ml/Kg + Insulin 1 u/5 gm glucose, or

•$NaHco_3$ amp 20 ml (17.5 mEq), if pt is acidotic, 1 amp over 5 min, or 1-2 ml/Kg (1-2 meq/Kg) or

•Normal saline + Lasix 20-80 mg, or 1 mg/Kg IV.

HYPERCALCAEMIA

Mild: Ca^+ level is 10.5 - < 12 mg/dl. Moderate: 12-14 mg/dl (think in malignancy if serum Ca^+ >13 mg/dl). Severe: if serum Ca^+ is >14 mg/dl.

98% of the body calcium is in the skeleton, only 2% is in the circulation of ẃ 50% is free calcium (ionized Ca^{++}) ẃ is the physiologically active & the reminder 1% is bound to proteins. The calcium regulation is under control of:- **1-PTH:** inhibits osteoblasts, stim-

ulate osteoclasts, thus stimulates bone resorption & renal tubular reabsorption of calcium, thus ⇧ blood calcium. **2-Calcitonin:** secreted by thyroid gland, inhibits osteoclasts, stimulate osteoblasts & ⇧ urine calcium excretion by inhibiting renal calcium reabsorption, thus ⇩ blood calcium.

Causes

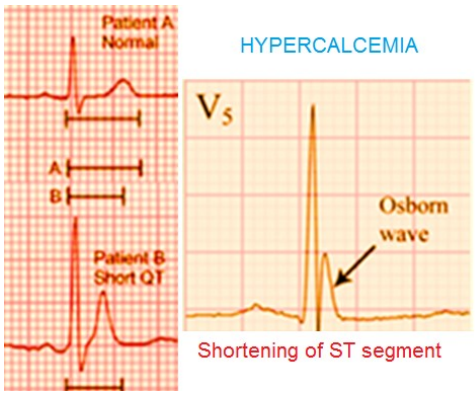
90% of hypercalcaemia are due to primary hyperparathyroidism & malignancies. The primary hyperparathyroidism include; parathyroid adenoma (90%), hyperplasia (5%) & parathyroid carcinoma (5%). Other causes include; iatrogenic from excess Vit D or excess dietary intake of calcium. May be caused by drugs as Thiazide diuretics, or Lithium or excess Vit A. Also é immobilization, hyperthyroidism, Milk alkali syndrome (result from use of more calcium for osteoporosis), Sarcoidosis, TB, Histoplasmosis, Paget`s disease of bone, Multiple myeloma & Bone metastasis.

Clinical picture

May range from non-existent to severe. Pt may present é stones (renal or biliary), bone or abdominal pain, polyuria, water depletion, dehydration, constipation, anorexia, nausea, weakness, lethargy, psychatric overtones (depression, confusion)& ECG changes.

ECG changes

•Shortening of QT interval. •J waves (Osborn wave) as notching of the terminal QRS, best seen in lead V5 (as é severe hypothermia). •Elevation of ST segment. • Arrhythmia.



Investigations

⇧ **PTH:** in primary, secondary, or tertiary hyperparathyroidism or é familial hypocalciuric hypercalcaemia.

⇩ **PTH:** in malignancy, excess Vit.D, granulomatous disease, milk alkali syndrome.

Serum Ca^+ level: pt is hypercalcaemic if serum ioniz ed Ca^+ **>** 5.6 mg/dl & total serum Ca^+ **>** 10.5 mg.

Urine Ca^+: if there is ⇧ of urine Ca think of hyperparathyroid, excess vit. D, or granulomatous disease. If urine Ca is ‹200 mg/day, think in familial hypocalciuric hypercalcaemia. If urine Ca is normal it is associated é milk alkali syndrome. In case of malignancy the urine Ca is variable (either ⇧, ⇩, or normal).

Urine phosphate: ⇧ in case of hyperparathyroidism, excess Vit D, or malignancy. It is normal in case of granulomatous disease, milk alkali syndrome or familial hypocalciuric hypercalcaemia.

Serum alkaline phosphatase: ⇧ in case of hyperparathyroidism, malignancy, granulomatous disease. ⇩ in case of excess Vit D. Normal in case of milk alkali sy & familial hypocalciuric hypercalcaemia.

Management

- Aggressive hydration: ½ normal saline or normal saline.
- Diuretic: no Thiazide diuretic to be given as it will ⇧ calcium level, loop diuretic as Furosemide to be given after rehydration.
- Calcitonin amp 50, 100u, 4u/Kg SC or IM.
- Bisphosphonates intravenous: act by reducing bone resorption.
- Steroids: useful in case of Vit D excess & granulomatous diseases.

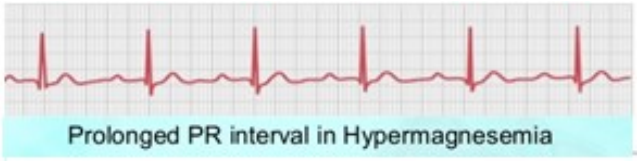
HYPERMAGNESAEMIA

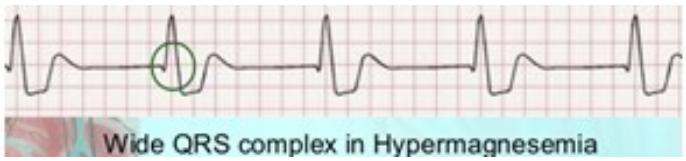
Clinical picture

Mg^{++} is required to support the heart muscle`s normal contraction-relaxation actions. ⇧ of Mg^{++} level may cause delayed conduction of electrical impulse. Too much ⇧ of Mg^{++} depress the CNS, breathing & produces Hypotension, ⇩ of heart rate, diaphoresis, flushing, drowsiness, ⇩ of the mental status, hyporeflexia, weakness, paralysis, heart block & ECG changes.

ECG changes

Prolonged PR interval, widened QRS, ventricular dysrhythmias & heart block.


Prolonged PR interval in Hypermagnesemia


Wide QRS complex in Hypermagnesemia

Causes

•⇧ Mg^{++} intake •⇩ Renal excretion •Dehydration •Addison's & other adrenal diseases •Hyperparathyroidism •Hypothyroidism •Kidney failure •Acidosis.

Diagnosis

▲ Mg^{++} is < 2.0 mEq/L & ▲ The ECG changes.

Management

•Diet restrictions for Mg. •Circulatory & respiratory support. •Administer diuretic, IV Lasix to ⇧ Mg excretion (only when renal function is adequate). •1 gm Ca gluconate reverses cardiac effects. •Haemodialysis may be used in severe cases of hypermagnesaemia because approximately 70% of serum Mg is not protein bound & can be removed through dialysis.

HYPERNATREMIA

Hypernatremia is much less common than Hyponatraemia & it is often iatrogenic (e.g. excessive normal saline), seen in 1% of hospitalized pt & is more common among infants & old age pts, risk factors ⇧ é age > 65 yrs & é dementia, mental or physical disabilities. Hypernatremia is defined as Serum Na **>** 145 mmol/l.

Classified into;

*Mild (Na 146-149).

*Moderate (Na150-169).

*Severe (Na ≥ 170).

⇧ Na^+ conc. in blood creates an osmotic gradient between the ECF (plasma, interstitial fluid) & ICF (cells fluid) causing movement of water into the extracellular space, in severe cases it affect brain cells causing shrinkage, permanent brain damage & IC Hge.

Causes

•Excess water loss. •Excess solute intake. •Hyperosmolar feeding. •Inadequate fluid intake. •Excess insensible water loss. •Tachypnea. •Burns. •Nephrogenic diabetes insipidus (deficient response of kidney to the ADH). •Central diabetes insipidus (deficient ADH secretion by posterior pituitary). •Diarrhea •Hypertonic dehydration •Osmotic diuretics as Mannitol •Hyperosmolar hyperglycemic stat •Iatrogenic as excess intake of normal saline or $Na^+Hco_3^-$ or é TPN. •Primary hyperaldosteronism •Acute & Chronic renal failure. •Drugs as Lithium & Amphotericin B.

Types

- Hypovolemic hypernatremia: H_2o deficit is **>** Na^+ deficit.
- Hypervolemic hypernatremia: excess Na^+ gain.
- Euovolemic hypernatremia.

Clinical picture

•Fatigue. •Nausea. •Vomiting. •Headache. •Flushed skin. •Confusion. •Seizures. •Coma & death. •⇧ Capillary refill time. •⇩ COP related to ⇩ of myocardial contractility.

Management

•Calculate water deficit: 0.6 X Wt X {(current Na ÷ 140) -1}

•Correction must be ve-very slowly & Na^+ should not be lowered by >2 meq/L/day, as over correction can lead to cerebral edema, seizures & death.

•The typical fluid is dextrose 5% in water.

HYPOKALAEMIA

As mentioned before, K^+ is vital for regulating the normal electrical activity of the heart. The ⇩ of the extracellular K^+ causes myocardial hyperexcitability é the potential to develop re-entrant arrhythmias. Nearly 98% of the body`s K^+ is intracellular. The normal serum level of k^+ is 3.5-5 meq/L. Hypokalemia divided into;

- Mild: K^+ 3-3.5 meq/L
- Moderate: K^+ 2.5-3 meq/L
- Severe: K^+< 2.5 meq/L.

Causes:

•Diarrhea/Laxative abuse.

•Vomiting / Metabolic alkalosis.

•Liver cirrhosis. DM. Primary Hyperaldosteronism.

•Steroids, Digitalis, ß-blockers, Thiazide, Loop diuretics.

•Renal tubular acidosis.

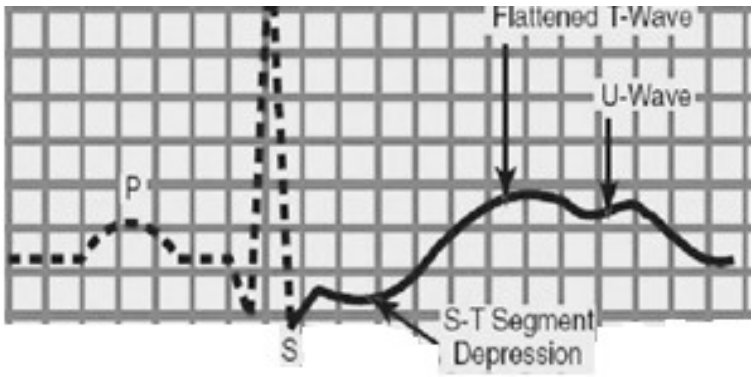
Clinical picture: symptoms depend upon the degree & duration of hypokalemia.

*Mild hypokalaemia are often asymptomatic.

*Moderate/severe hypokalaemia present é; Cardiac arrhythmia or arrest. Paralytic ileus. Muscle weakness/cramps & Polyuria.

ECG changes

Hypokalemia
ECG tracing has ST-segment depression, flattened T-wave, and a U-wave.



•Prolonged P-R interval. •Widened QRS complex •QT interval usually indiscernible as T-wave flattens. •Depressed S-T segment. •T wave amplitude is ⇩, inverted T wave & U wave may be seen.

Management

- Mild hypokalemia treated by oral kcl tab maximum 20 meq (2 tab of 600 mg Kcl)
- Severe form treated by Kcl 10 ml amp 15% (contain 1.5 gm Kcl), added to NS & infused over 12-24 hrs, maximum dose not more than 40 meq/L of NS. Repeat until all symptoms resolve.

HYPOCALCAEMIA

Ca is important in cardiac function; it exerts a +ve inotropic effect on heart. ⇩ in Ca levels may cause ⇩ myocardial contraction. Hypocalcaemia is defined as total serum Ca^{+} < 8.5 mg/dL or ionized Ca^{+} < 4.6 mg/dL. The total serum Ca^{+} levels must be corrected for serum albumin levels. Ionized Ca^{+} levels do not require such correction. Acute, severe hypocalcaemia is a medical emergency. Approximately 40% of serum Ca^{+} is ionized (free),

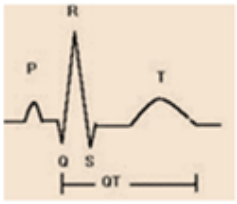
while the other 60% is complexes, primarily to albumin. Only the ionized Ca^{+} is transported into cells & metabolically active. ⇩ in the ionized Ca^{+} cause symptoms. Hypoalbuminemia alters total serum Ca^{+} conc. éout affecting the ionized Ca^{+}. The serum total Ca^{+} conc falls approximately 0.8 mg /dL for every 1 gm/dL reduction in the serum albumin conc.

Causes

Major factors that influence the serum Ca^{++} conc. are PTH, Vit D & serum phosphorus level. Hypocalcaemia most commonly occur é Vit D deficiency, chronic renal failure, hypoparathyroidism (typically due to neck surgery, rarely from autoimmune destruction or congenital abnormality) or may be idiopathic hypocalcaemia.

ECG changes

Prolonged QT Interval

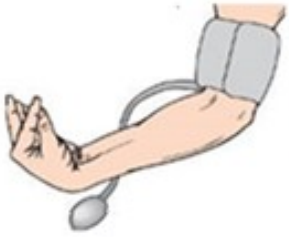


•Prolonged Q-T interval. •Prolonged S-T segment. •May associated é flattening of T wav

Clinical picture

A. Trousseous sign: finger taping over aathe course of facial nerve will result in twitching of muscles of the face

B. Convulsion- Carpopedal spasm



C. Chvosteck sign: keeping SBP for 3 min. will result in carpopedal spasm

Cardinal features are •Muscle spasm (Trousseous sign, Chvosteck sign, Carpopedal spasm) •Irritability •Tetany •Paraesthesia •Seizures & •Arrhythmias.

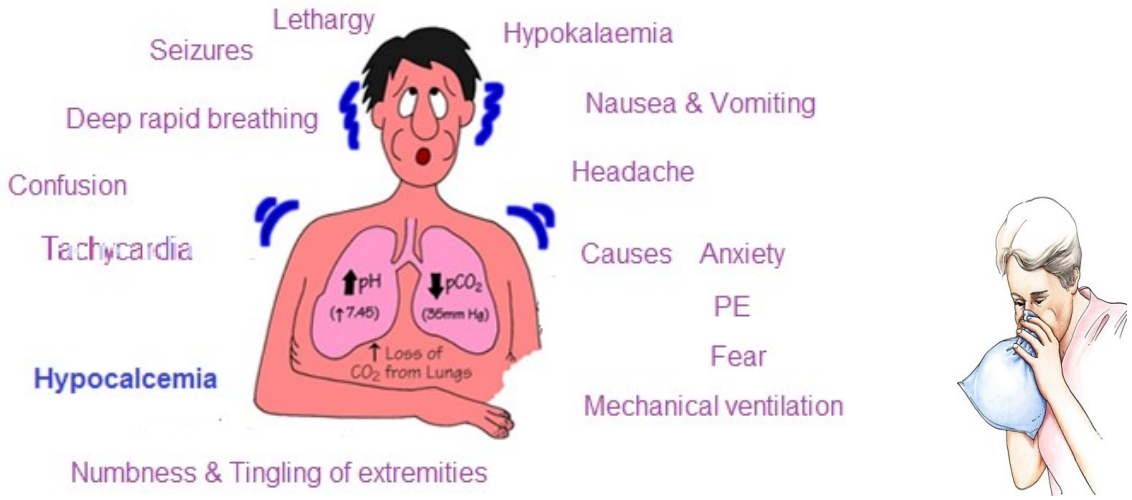
Investigations

- ⇧ PTH.
- ⇩ Ca & ⇧ Phosphorus levels in blood.
- Normal serum alkaline phosphatase.
- X Ray skull (basal ganglia calcification).

Management

Ca gluconate 10% ampule 10 ml=I gm=4.5 meq, 100 mg or 1 ml/Kg stat, slowly & strictly intravenous. Chronic hypocalcaemia often responds to Rx é vit. D derivatives & calcium.

HYPERVENTILATION SYNDROME



Over breathing, breathing is fast & deeper, result in inhalation of lot of O_2 & ⇩ of Co_2 in blood. Follow psychic trauma. More common in women, anxiety, fear & mechanical ventilation.

Clinical picture

Tachypnea, irritability, carpopedal spasm, spasm of neck & jaw muscles, result from alkalosis &excess washing of CO_2 causing respiratory alkalosis & hypocalcemia.

Management

- Breathing through a Paper Bag.

•Valium 5, 10 mg amp, 0.25 mg/ Kg/dose IV/IM stat (suppository 5, 10 mg), then daily dose of 0.25 mg/Kg/ day ÷ 4

•Ca gluconate 10%, 10 ml ampule, strictly IV 1 ml/Kg =100 mg/Kg, after dilution é 5% Glucose in a ratio of 1: 4 é monitoring of heart rate.

•Treating alkalosis is thro-ugh giving normal saline & correction of hypokalaemia.

HYPOMAGNESAEMIA

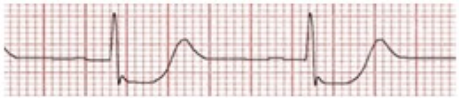
(Magnesium ˂1.4 meq/L)

Causes

Mg^{++} deficiency is often associated é low blood Ca &/or K. Hypomagnesaemia may be caused by: •Irritable Bowel Sy. •Ulcerative Colitis - because Mg^{++} is absorbed in the intestines & then transported through the blood to cells & tissues. •Alcoholism or withdrawal from alcohol. •Malnutrition. •Starvation. •Inadequate intake of mineral •Kidney disease. •Pancreatitis. •Hyperglycemia. •Medications- as Diuretics (⇧ loss of Mg^{++} through urine), Digitalis, Cisplatin, Cyclosporine, Aminoglycoside antibiotics, insulin administeration. •Large amount of Mg^{++} can be lost by prolonged exercise. •Excessive sweating. •Chronic diarrhea. •Hypoparathyroidism as PTH helps control Ca^{++}, Ph^{++}, Mg^{++} & Vit. D levels within the blood & bone, in such condition the blood Ca levels fall & Ph levels rise.

Clinical picture: ▲Anorexia. ▲Nausea. ▲⇩ Intestinal motility. ▲+ve Trousseau's & Chvostek's signs, ▲Neuromuscular irritability & Hyperreflexia. ▲Seizures. ▲Shallow respiration. ▲Laryng ospasm. ▲Hyperventilation. ▲Cardiac arrhythmia. ▲Apathy. ▲ Mood changes. ▲Agitation. ▲Confusion. ▲Insomnia.

ECG changes



•Depressed ST segment. •Tall T waves. •Irregular ventricular rhythm.

Management

•Eliminate contributing drugs.

•Seizure precautions.

•IV $MgSO_4$, 25-50 mg/kg/dose for replacement & 30-60 mg/kg/day for maintenance, IV dose must be given over 120 min via IV pump (500 mg $MgSO_4$ = 49.3 mg elemental Mg = 4.06 meq Mg). Monitor hourly $MgSO_4$. •Diet Therapy.

HYPONATRAEMIA

(Serum Na^+ <135 meq/L)

Na^+ is the main electrolyte in the body & is important for many body functions, its normal value in blood is 135-145 meq/L. Hyponatraemia represent relative excess of water in relation to Na^+. Hyponatraemia is the most common electrolyte abno- rmality in geriatric & hospitalized pt, seen in 30% of pts treated in the ICU & in 3% of hospitalized pts, swelling of the body is the most danger effect of Hyponatraemia causing brain oedema, seizures, coma & respiratory arrest.

Causes

•⇧ Water retention. •Low salt intake. •Polydipsia. •Advanced renal failure. •Thiazide diuretics. •Adrenal insufficiency.

Types

✶***Hypovolemic hyponatraemia;*** ⇩ Na^+ & H_2O (signs of dehydration).

✶***Hypervolemic Hyponatraemia;*** ⇩ Na^+& ⇧ H_2O (oedema, liver cirrhosis, HF).

✶***Euvolumic Hyponatraemia;*** normal appearance.

Clinical picture

*Mild Hyponatraemia (Na^+ **<**130 meq/l); malaise, nausea, drowsiness.

*Moderate Hyponatraemia (Na^+ ﹤120 meq/l); vomiting, headache, confusion, muscle

cramps, ⇩ reflexes. *Severe Hyponatraemia (Na^+ ⟨110 meq/l); seizures, coma.

Management

*Serum & urine osmolality & urine Na^+ conc are initial tests to do.

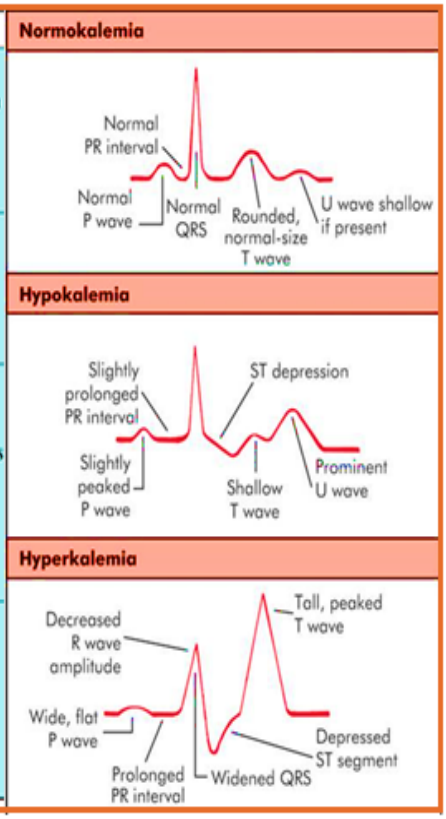
*Mild or no symptoms: fluid restriction.

*Severe symptoms: give bolus of 3% saline, 100 ml (this raise Na^+ level 2-3 meq/l). Calculate Na^+ deficit (total body water X (Desired Na^+ - Pt Na^+). The total body water is equal to 60% of body weight in males & 50% in females). Correction should not exceed 10 meq/L during the 1st 24 hrs to avoid central pontine myelinolysis ẃ is a form of osmotic demyelination in ẃ the symptoms start to occur after 2-6 days from the rapid correction in the form of dysarthria, dysphagia, lethargy, quadriparesis, seizures & coma.

Example; 60 Kg female é serum Na^+ level 116 meq/l.? We need in the 1st 24 hrs to raise serum Na^+ level to 124 meq. ∴Na^+ deficit = 50% X 60 X (124 ÷ 116) =240 meq. ∴ Give 240 meq Na^+ during the next 24 hrs. ∵ 3% saline solution contains Na^+ 500 meq/l (hypertonic saline) & the 0.9 saline contain Na^+ 154 meq/l (normal saline). # Give 480 ml of the hypertonic saline or 1560 ml of the normal saline over the next 24 hrs & Repeat until all symptoms resolve.

SUMMARY

Electrolyte	Excess	Deficit
Sodium (Na)	Hypernatremia Thirst CNS deterioration Increased interstitial fluid	Hyponatremia CNS deterioration
Potassium (K)	Hyperkalemia Ventricular fibrillation ECG changes CNS changes	Hypokalemia Bradycardia ECG changes CNS changes
Calcium (Ca)	Hypercalcemia Thirst CNS deterioration Increased interstitial fluid	Hypocalcemia Tetany Chvostek's, Trousseau's signs Muscle twitching CNS changes ECG changes
Magnesium (Mg)	Hypermagnesemia Loss of deep tendon reflexes (DTRs) Depression of CNS Depression of neuromuscular function	Hypomagnesemia Hyperactive DTRs CNS changes



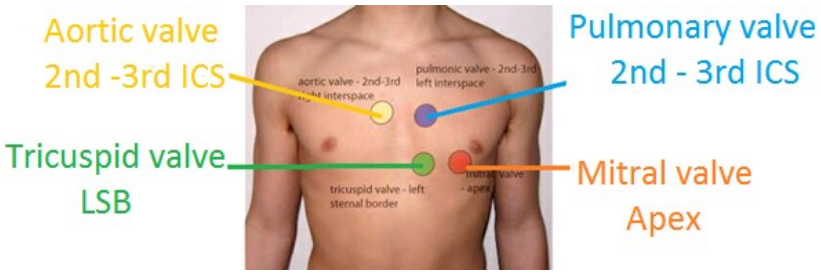
INTRAVENOUS FLUID COMPARISON

Type	Solution	Uses	Special Considerations
Isotonic	Dextrose 5% in water	Fluid loss. Dehydration. Hypernatremia	Use cautiously in renal & cardiac pt. Can cau-se fluid overload
Isotonic	0.9% NaCl	Shock. Hyponatraemia. Blo-od transfusion.R esuscitation. Fluid challenges. DKA	Can lead to overload. Use é caution in pt é HF or oedema
Isotonic	Ringer`s Lact.	Dehydration. Burns. Lower GI fluid loss Acute blood lo- ss. Hypovolemia	Contain K^+, Don`t use é renal failure. Don`t use é liver disease, can`t metabolise lactate
Hypotonic	0.45% NaCl	Water replacement DKA. Gastric fluid loss from NG tube or vomiting	Use é caution. May cause cardiov-ascular colla-pse or ⇧ IC pressure. Don't use é liver disease, or burns
Hypertonic	Dextrose 5% In ½ NS	Later in DKA Rx	Use only when blood sugar falls < 250 mg/ dl
Hypertonic	dextrose 5% in NS	Temporary Rx for shock if plasma expanders not available. Addison's crises	Do not use in cardiac or renal pt
Hypertonic	Dextrose 10% in Water	Water replacement TPN	Monitor blood sugar level

	Na (mEq/L)	K (mEq/L)	Cl (mEq/L)	HCO3 (mEq/L)	Dextrose (gm/L)	mOsm/L
D5W					50	278
½ NS	77		77			143
D5 1/2NS	77		77		50	350
NS	154		154			286
D5NS	154		154		50	564
Ringers Lactate (RL)	130	4	109	28	50	272

PRACTICAL POINTS

Sites of heart valves



Heart Sounds

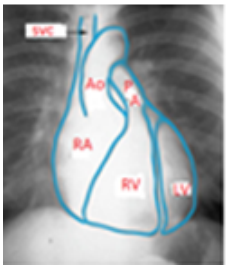
*First heart sound (S1): result from closure of mitral & tricuspid valves.

*Second heart sound (S2): may be soft, loud, widely split, or reversed split.

*Third heart sound (S3) : diastolic filling of the ventricle. It is normal persons < 30 yrs old. Characteristic of LV failure.

*Fourth heart sound (S4): Atrial contraction against a stiff ventricle. May be heard in AS, HOCM, hypertension.

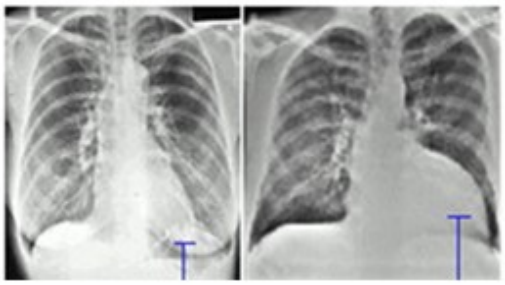
*Murmurs



opposite	Stenosis	Regurge
Aortic	Systolic	Diastolic
Pulmo	Systolic	Diastolic

- Ejection systolic murmur: AS or PS
- Soft systolic murmur: ASD, or VSD.
- Diastolic murmurs: AR or PR.

CXR



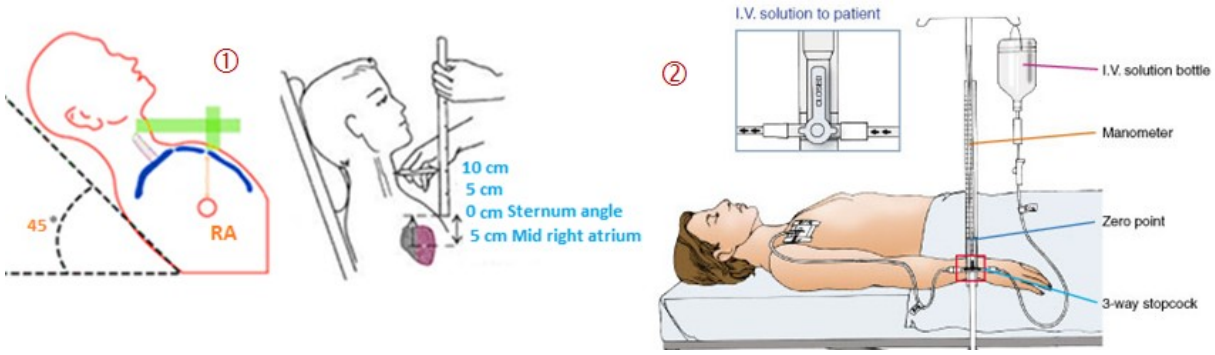
The X Ray on the left shows a normal heart, on the right, the heart is enlarged(Rt)

- RVH: cardiac shadow ⇧, no angle between apex & diaphragm.
- LVH: cardiac shadow ⇧, angle between apex & diaphragm.

Causes of wide pulse pressure

▲Anaemia ▲Thyrotoxicosis ▲AR ▲Liver disease ▲Systolic hypertension (old age)

Central Venous Pressure



CVP, also known as mean venous pressure' (MVP) is the pressure of blood in the thoracic vena cava, near the right atrium of the heart. CVP reflects the amount of blood returning to the heart & the ability of the heart to pump the blood into the arterial system. The normal range of CVP is 5-10 cmH_2o when taken from the mid axillary line at the 4^{th} ICS, & equal to 8-15 cmH_2o for pt on ventilators.

Method for measuring CVP

① Indirect assessment: inspection of jugular vein pulsation in neck.

② Direct assessment: fluid filled monitor connected to CVC, calibration transducer

③ Electronic transducers (in ICU).

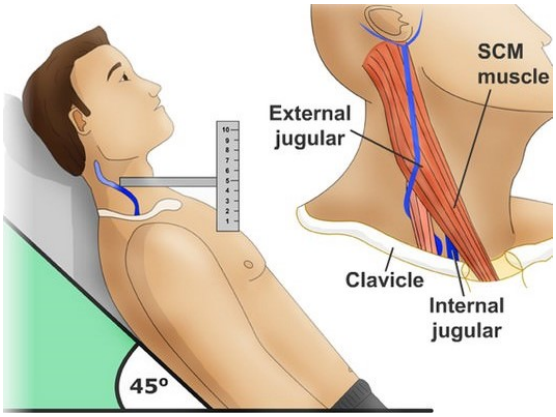
Position the pt at 45^0 angle on the examining table. Place ruler at the sternal angle

(the sternal angle is about 5 cm above the right atrium). Hold another ruler horizontally at the top of the venous pulse & note how many cm this is above the sternal angle. Add 5 cm to this total (accounting for the distance between the sternal angle & Rt atrium), the total is the JVP. Normal is **<** 9 cm. The normal range of CVP is 5-10 cmH_2o when taken from the mid axillary line at 4^{th} ICS & equal to 8-12 cmH_2o for pt on ventilators. indicator of volume overload, especially on the Rt side of the heart. Best combination of sensitivity & specificity in diagnosis of HF.

Indications for CVP monitoring

- Emergency fluid resuscitation.
- Hemodialysis.
- TPN.
- Administration of irritant drugs.
- Poor venous access.

Jugular Venous Pulse



Defined as the oscillating top of vertical column of blood in right internal jugular vein that reflects pressure changes in Right Atrium in cardiac cycle. Jugular venous pulse is defined as the oscillating top of vertical column of blood in the right Internal Jugular Vein (IJV) that reflects the pressure changes in the right atrium in cardiac cycle. Jugular venous pressure (JVP) is the vertical height of oscillating column of blood.

Why is Internal Jugular Vein preferred?

- IJV is anatomically closer to and has a direct course to right atrium while External Jugular vein does not directly drain into Superior vena cava.
- IJV is valveless & pulsations can be seen. Due to presence of valves in External jagular vein, pulsations cannot be seen.
- Vasoconstriction secondary to hypotension (as in CHF) can make EJV small & barely visible. •EJV is superficial and prone to kinking.

Why is Right Internal Jugular Vein preferred?

- Right jugular veins extend in an almost straight line to superior vena cava, thus favoring transmission of the hemodynamic changes from the right atrium.
- The left innominate vein is not in a straight line & may be kinked or compressed between Aortic Arch & sternum, by a dilated aorta, or by an aneurysm.

Evaluation of JVP

•Level. •Waveform. •Respiratory variation in level and wave pattern. •Hepatojugular reflux. •Venous hum. •Liver size & pulsations

Technique of measuring JVP

1) Position: Semi-reclining position ē 45° angle between the trunk (not the neck) & the bed. then, turn the head slightly towards left shoulder, so that the neck muscles are relaxed. Not in sitting position: because the upper level of venous column is below the clavicle. Not in supine position: because the whole venous column moves beyond the angle of jaw into the intracranial cavity.

2) Identify Jugular venous pulsation

- Assure good lighting (can use tangential beam of light through torch).
- Look between the two heads of sternocleidomastoid muscle.

- Note the upper level of pulsation, waveform & respiratory variation.

Do not mistake the carotid pulsations for venous pulsations.

JUGULAR VEIN	CAROTID ARTERY
No pulsations palpable.	Palpable pulsations.
Pulsations obliterated by pressure above the clavicle	Pulsations not obliterated by pressure above the clavicle
Level of pulse wave decreased on inspiration; increa-sed on expiration.	No effects of respiration on pulse.
Usually 2 pulsations per systole (x & y descents).	One pulsation per systole.
Prominent descents.	Descents not prominent.
Pulsations sometimes more prominent ē abdominal pressure.	No effect of abdominal pressure on Pulsations

3) Locate the sternal angle (Angle of Louis):

It can be felt as a transverse prominence, about 5cm below the suprasternal notch at the level of 2nd costal cartilage.

4) Measurement

Measure the vertical distance (cm) between the horizontal lines drawn from the upper level of venous pulsation & the sternal angle. This can be done by using 2 rulers – one placed horizontal to the upper level of pulsation & another taking the vertical distance of that ruler from the sternal angle.

5) Calculate the right atrial pressure

Normally, the centre of right atrium is 5 cm below the sternal angle. Hence, Add + 5 cm to the above measurement to obtain the right atrial pressure.

6) Conversion: 1.3 cm of H2O or blood = 1 mmHg

Interpretation

1) Normal level of JVP:•From sternal angle: <4 cm.•From centre of right atrium: <9cm •In mmHg: <7 mmHg

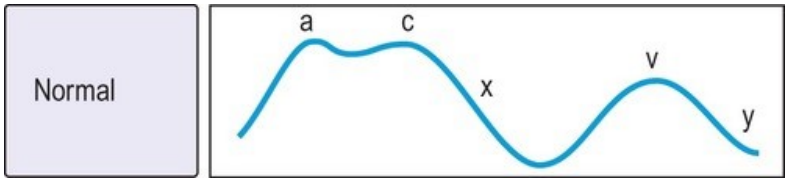
Causes of elevated JVP (Jugular venous distension):

1- RVF. 2- Pericardial compression (constriction/tamponade). 3- Tricuspid stenosis. 4- Superior vena cava obstruction- no pulsations. 5- Excessive fluid administration. 6- Renal failure. 7- ASD ē mitral valve disease.

Causes of lowered JVP : 1-Dehydration. 2-Hypovolemia.

2) Wave pattern & Abnormalities

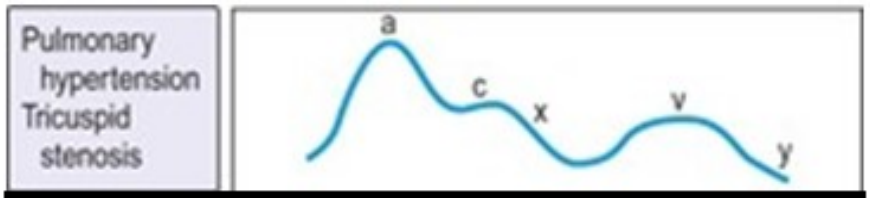
The normal JVP consists of 3 ascents or positive waves (a,c and v) &2 descents or negative waves (x, x' & y)



a wave is produced by atrial systole. x =atrial contraction finishes.

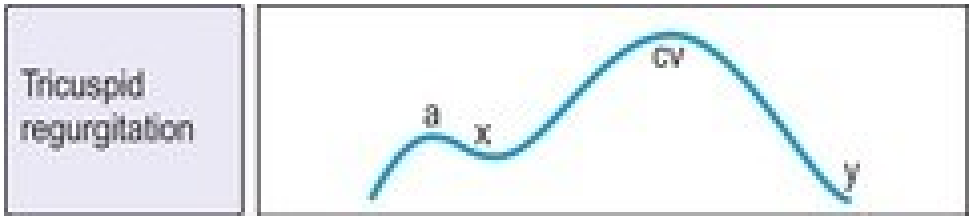
c wave occurs during the x descent & is due to transmission of right ventricular sy-stolic pressure before the tricuspid valve closes. v wave = venous return filling the right atrium. The y descent follows the v wave when the tricuspid valve opens.

Increased a wave occurs in what conditions?



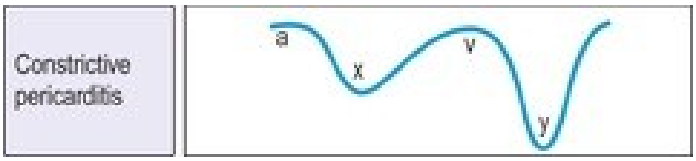
Right ventricular hypertrophy 2ry to pulm. hypertension or pulmonary valve sten-osis. Giant cannon waves occur in complete heart block & ventricular tachycardia.

Giant v waves occur in?



Giant v waves occur in tricuspid regurgitation.

A steep y decent is seen in?



A steep y descent is seen in constrictive pericarditis & tricuspid incompetence.

3) Venous hum

Continuous bruit over neck veins (normally noiseless) due to ⇧ velocity of blood flow or ⇩ viscosity of blood. May be;

- Physiological: Children, Pregnancy.
- Pathological: Hyperkinetic states, Anaemia, Thyrotoxicosis, Beriberi, Intracranial AV fistula.

4) Respiratory variation

Normal: venous column rises during expiration & falls during inspiration.

Kussmaul's sign: Paradoxical rise in JVP during inspiration seen in case of; Constrictive pericarditis, Cardiac tamponade, Restrictive cardiomyopathy (because of ⇧ venous return in inspiration cannot be accommodated as increased pulmonary filling leading to inspiratory filling of neck veins.

5) Hepatojugular reflux (Provocative test)

When pressure is applied over the liver by pressing firmly below the right costal cartilage margin for 30 sec, the venous pressure gets exaggerated initially due to ⇧ venous return.

Later the myocardium accomodates the extra venous return & the level falls. Positive test: rise in JVP for >10 sec. in case of:-

- Early HF.
- False positive: Valsalva (abdominal guarding), fluid overload.
- False negative: in case of:- SVC/IVC obstruction. Budd Chiari syndrome.

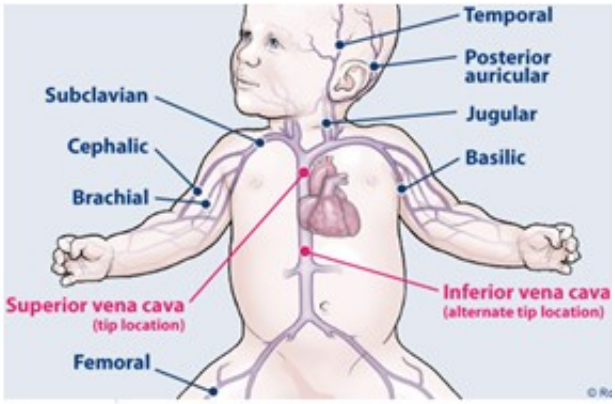
This test also helps to differentiate venous pulsation from the arterial pulsation.

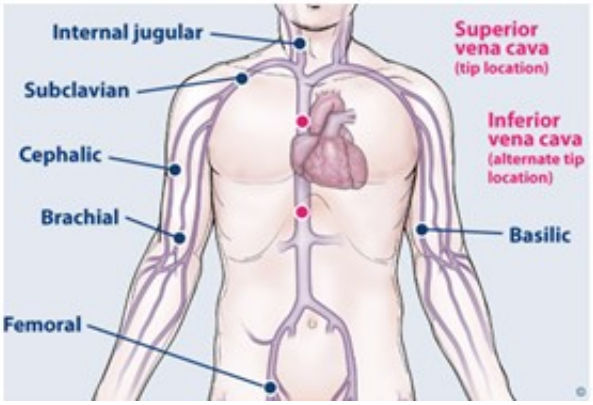
6) Liver pulsations

In an infant, the liver is the only guide to the recognition of elevated right atrial pr-essure as the JVP is difficult to delineate.

Central lines Canulation

Ultra sound guide for CVC

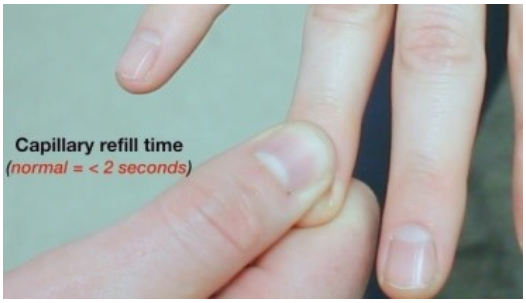




Capillary refilling test

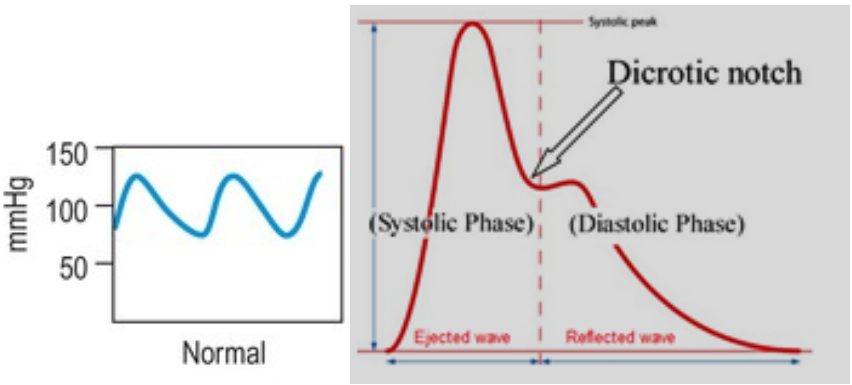
Crude method of assessing blood circulation, in infants can tested by applying pressure over the sternum, or foot. In adults can be tested in finger nails while elevated above the

level of the heart, or in middle of forehead. Restoration of time > 3 sec is considered sluggish circulation & means that pt need expansion of intravascular volume, seen in case of shock, dehydration, ⇩ COP, or hypothermia.



PULSE

One of the important vital signs, & may be the only available clew for diagnosis of serious illness ẃ is going on when other data not available? Pulse is the indirect measure of heart beat & activity of the heart. It is a wave of expansion & recall occurring in an artery in response to the pumping action of heart. The normal pulse has a small anacrotic wave on the upstroke ẃ is not felt. This is followed by a big tidal or percussion wave ẃ is felt by the palpating finger. On the following down stroke there is a notch (dicrotic notch) followed by a wave (dicrotic wave) both of ẃ are not normally palpable. In adults, the normal pulse appears at regular intervals & has a rate between 60-100 bpm. There may be a mild variation in the rate between the two phases of respiration ẃ is called sinus arrhythmia.



Procedure of taking pulse

Place tips of 2 fingers other than thumb lightly over pulse site. Thumb is not used in assessing pulse as it has its own ẃ can be mistaken for pt`s pulse. Do not press the artery ē more force. After getting the pulse regularly, count the pulse for one whole minute looking at the second hand on the wrist watch. Assess for rhythm, & volume of pulse & condition of blood vessel.

Characteristics of pulse

1. Pulse rate: number of beats per minute. Normally in adults it is 60-100 bpm.
2. Rhythm: is the time interval between pulse beats. Normally are equal & regular.
3. Tension: is the degree of compressibility & depends upon the resistance of the wall of the artery.
4. Volume: it is the fullness of artery. It is force of blood felt at each beat.
5. Delay: for both radial pulses, & radial & femoral pulses.

Purposses of taking pulse

-To establish base line data.

-To check abnormalities in rate, rhythm & volume.

-To monitor any changes in health status of the pt.

-To assess response of heart to cardiac medications, activity, blood volume.

-To check the peripheral circulation.

-To determine number of heart beat per minute.

Factors affecting the pulse

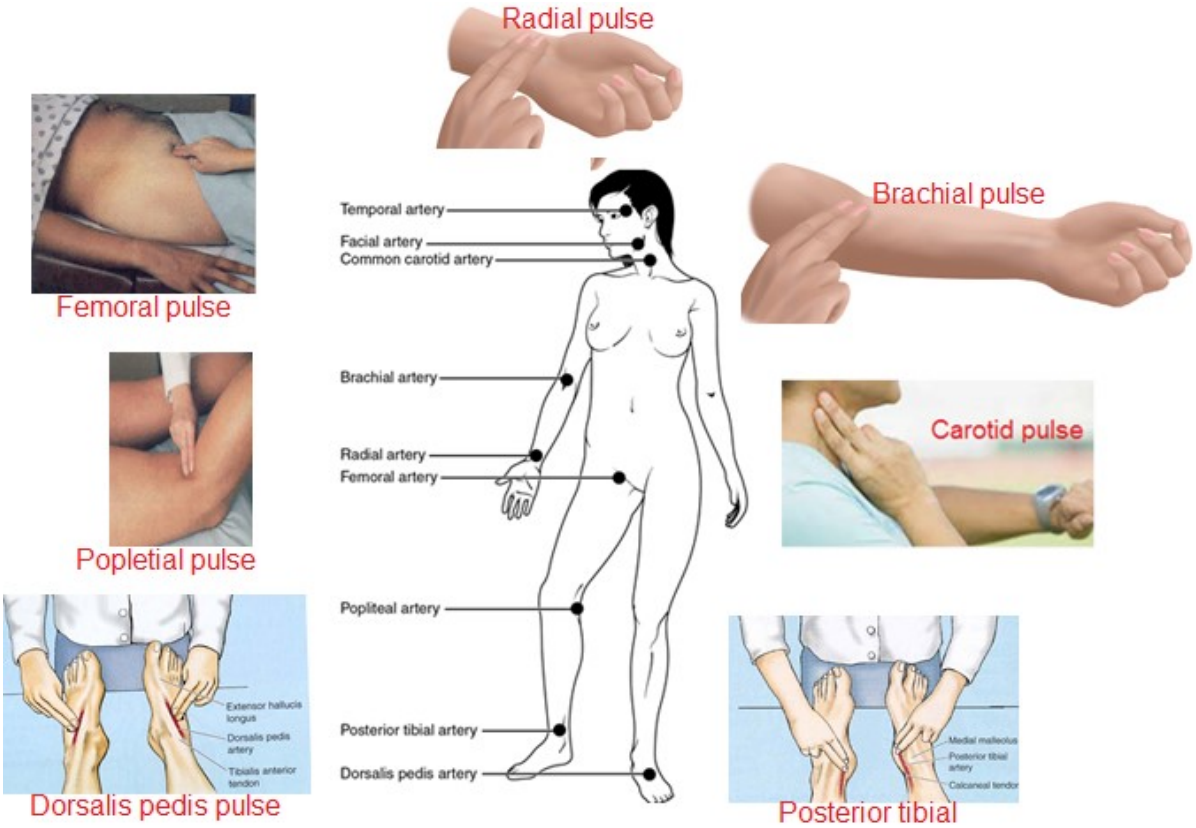
- Age: very old person have slow pulse rate & children will have faster beat.
- Sex: females have a slightly higher pulse rather than males.

- Exercise/activity: pulse rate is much faster during exercise.
- Stature: the short & thin persons have a more rapid pulse than tall & heavy.
- Emotions: anger or excitement ⇧ the pulse rate temporally.
- Fever: when body temp ⇧, the pulse rate usually ⇧ as well. Pulse ⇧ at a rate of about 10 bpm each degree rise of body temp.
- Blood pressure: when BP ⇩, the pulse may ⇧ to ⇧ the flow of blood. If BP ⇧, the pulse rate may ⇩ to correct the blood flow.
- Drugs: stimulant drugs ⇧ the pulse rate. Depressant drugs ⇩ the heart rate.
- Disease condition: heart disease, thyroid disease & other infections affect pulse.
- Acute pain & anxiety: ⇧ pulse rate.
- Sever & chronic pain: ⇩ pulse rate.
- Position: ⇩ while lying & ⇧ while standing.
- Hypovolemia/Hemorrhage: blood loss ⇧pulse rate because of demand of O_2.

Normal Pulse Rates

Age	Rate
Before birth	140–150
At birth	90–160
First year of life	115–130
Childhood years	80–115
Adult	60–80

Sites of checking pulse



❄ Radial: inner aspect of the wrist on thumb site.

❄Temporal: over the temporal bone or superior & lateral to the eye.

❄Carotid: at the side of the trachea where the carotid artery runs between the tr-achea & sternocleidomastoid muscle.

❄Apical: left side of the chest in the 4^{th}, 5^{th}, or 6^{th} ICS in the MCL.

❄Brachial: medially in the antecubital space, above the elbow.

❄Femoral: below inguinal ligament, midway between symphysis pubis & ASIS .

❄Popliteal: medial or lateral to the popliteal fossa ē knees slightly flexed.

❄Posterior tibial: on the medial surface of the ankle behind the medial malleolus.

❄Dorsalis pedis:along dorsum of foot between extensor tendons of 1^{st} & 2^{nd} toe.

❄Facial: at the outer angle of the lower jaw.

Abnormal Pulses

Tachycardia: when the resting pulse rate ⇧ to > 100 bpm in adult.

*Sinus: •Exercise •Infants •Excitements •Anxiety •Fever •Hyperthyroidism.

*Medication: •Ca Ch BL (Nifedipine) •Sympathomimetics •Vasodilators.

*Arrhythmia: •Atrial Fibrillation •Atrial flutter •Ventricular tachycardia.

Bradycardia: a pulse rate < 60 bpm.

*Sinus rhythm: •Sleep •Athletic training •Hypothyroidism.

*Medication: •Beta-blockers •Digoxin •Verapamil, Diltiazem.

*Arrhythmia: •Carotid sinus hypersensitivity •Sick sinus syndrome •2nd degree heart block. Complete heart block.

Causes of irregular pulse

*Occassionaly Irregular pulse: Extrasystole.

*Regular irregularity: •Ectopic beat occurring at a regular interval •2nd degree heart block • Sinus arrhythmia.

*Irregular irregularity pulse: •Atrial fibrillation •Multiple ectopics.

Pulse apex deficit

Difference in heart rate & pulse rate;

• Atrial fibrillation(> 10/ min)

•Multiple ectopics (< 10/min)

High volume pulse

Physiological causes: •Exercise •Pregnancy •Advanced age •⇧ environment temp

Pathological: • Arteriosclerosis •Exercise •AR •PDA •Atriovenous fistula •Fever.

•Thyrotoxicosis •Anaemia •Beri Beri •Complete heart block •Cirrhosis liver.

Low volume pulse

Causes: •LVF. •Hypovolemia. •Peripheral arterial disease. •Shock. •Severe aortic stenosis. •Pericardial effusion.

Varying volume

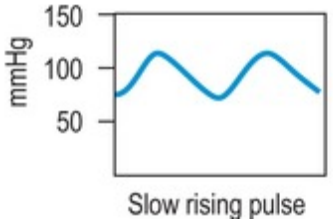
•Combination of low, normal or high volume pulse in varying manner.

•Seen in: Atrial fibrillation or Ventricular tachycardia.

Thready pulse

Pulsations are not easily felt & slight pressure causes it to disappear. The pulse rate is rapid & the pulse wave is small & disappears quickly. This is seen in shock.

Weak pulse / Pulsus Parvus



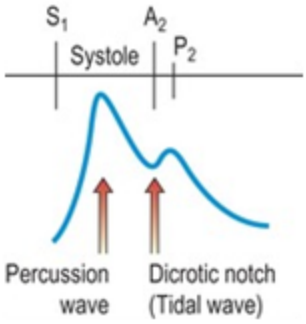
The pressure is diminished & the pulse feels weak & small, reflecting ⇩ stroke volume (e.g. HF), restrictive pericardial disease, hypovolemia, MS & ⇧ peripheral resistance (e.g. exposure to cold, severe CHF). Pulsus Parvus (weak & delayed) is seen in AS. The pulse is stronger than thready, light pressure causes the pulse to disappear.

Anacrotic Pulse

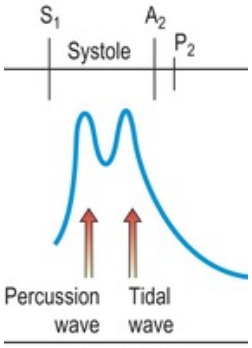
Is a slow rising, double beating pulse where both the waves are felt during systole. The waves that are felt are the anacrotic wave & the tidal wave. It is best felt in the carotids in aortic stenosis.

Dicrotic pulse

Description: Occurs in? results from an accentuated dicrotic wave. It occurs in sepsis, hypovolaemic shock & after aortic valve replacement.



Pulsus Bisferiens



Is a rapid rising, twice beating pulse (⇧arterial pulse ē a double systolic peak), where both the waves are felt during systole. Here the percussion wave is felt first followed by a small wave. It is seen in:

- Idiopathic hypertrophic subaortic stenosis (HCM).
- Severe AR.

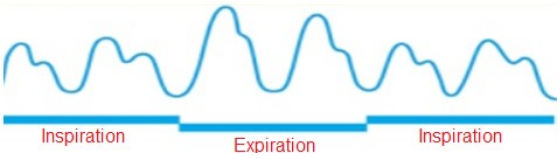
Pulsus Alternans

Variation in pulse amplitude occurring ē alternate beats due to changing systolic pressure (Change in ventricular contractility, causing changes in end-diastolic volume & pressure). When the cuff pressure is slowly released while taking BP, phase I Korotkoff sounds are initially heard only during the alternate strong beats; ē further release of cuff pressure, the softer sounds of the weak beat also appear. Degree of pulsus alternans can

be quantitated by measuring the pressure difference between the strong & weak beat.

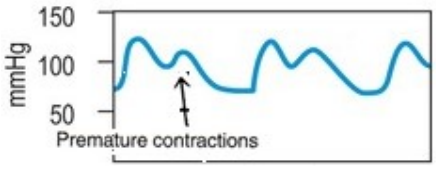
Causes: •LVF – usually accompanied by a left-sided S3 •May be seen in severe AR.

Pulsus Paradoxus



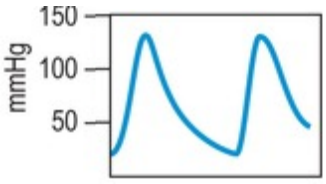
Pulse becomes smaller or disappear at end of deep inspiration (normally). It called Paradoxus pulse as heart sounds still be heard on auscultation over precordium, when no pulse is palbable at radial artery. SBP normally falls by 3-10 mm during inspiration. This is because though there is ⇧ venous return to the right side of the heart there is relative pooling of the blood in the pulmonary vasculature as a result of lung expansion & more negative intrathoracic pressure during inspiration. This ⇩ the venous return to the left atrium &ventricle & subsequently causes fall in left ventricular output decreasing the arterial pressure. When the SBP falls > 10 mmHg during inspiration, it is referred to as pulsus paradoxus. A reverse pulsus paradoxus may occur in pts receiving continuous airway pressure on a mechanical ventilator. Pulsus paradoxus is seen in superior vena cava obstruction, lung conditions like asthma, emphysema or airway obstruction, cardiac conditions like pericardial effusion, constrictive pericarditis & severe CHF.

Pulsus Bigeminus

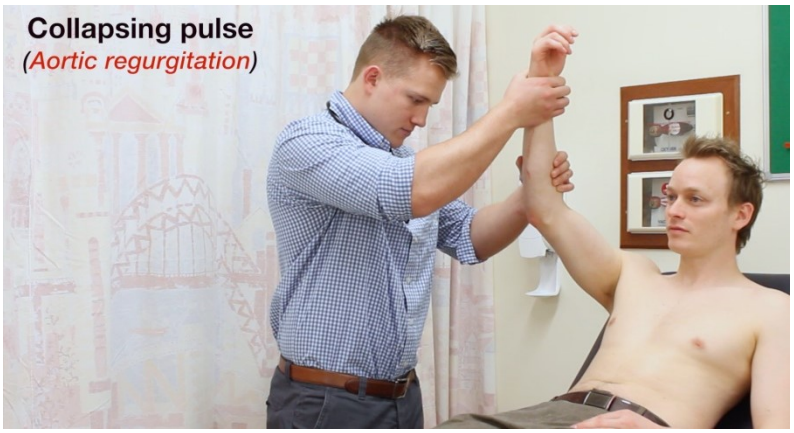


Normal beat alternating ē a premature contraction. May masquerade as pulsus alter-nans. Causes include; ⇩ BP (e.g. severe HF, hypovolemic shock, cardiac tamponade) & peripheral resistance (e.g.fever). Aortic valve replacement. Present in normal individuals after exercise.

Collapsing' or 'water hammer' pulse



A 'large-volume pulse characterized by a short duration ē a brisk rise & fall. This is be-st appreciated by palpating the radial artery ē the palmer aspect of four fingers while elevating the pt's arm above the level of the heart. Is associated ē ⇧ stroke volume of the left ventricle & ⇩ in the peripheral resistance, leading to a wide pulse pressure. The pulse strikes the palpating finger ē a rapid, forceful jerk & quickly disappears. It is caused by the artery suddenly emptying because some of the blood flows back from the aorta into the ventricle. It may be seen in fever, alcohol consumption & pregnancy It is also seen in high output states like anemia, beri beri or cor pulmonale, liver cirrhosis, Paget's disease, AV fistula, thyrotoxicosis. Cardiac lesions like AR, rupture of sinus of Valsalva into the heart chambers, PDA, aortopulmonary window & systolic hypertension may show Water hammer pulse as well. A collapsing pulse is characteristic of aortic valvular regurgitation or a persistent ductus arteriosus.



CONGENITAL HEART DISEASES

CHD is a heart problem that`s present at birth caused by improper development of the heart during fetal development.

Incidence: 1% of babies are born ē CHD, 30% of them require intervention to prevent death in the 1st year of life. 90% of cases have no known cause while 5% of cases are related to chromosomal abnormality & 2% are related to environmental factors. It is cyanotic in 22 % & acyanotic in 78 % of cases.

Simple way to classify Congenital Heart Diseases.

- Acyanotic (Left to right shunt): VSD, PDA, ASD.
- Cyanotic (right to left shunt): the 5 " T_s": T4, TGA, TA, Ta, TAPVR.
- Obstructive: AS, PS, COA.

Commonest group of life threatining Congenital Heart Diseases.

VSD (30:50%) -PDA (10%) -ASD (6%) -PS (6%) -CoA (6%) -AS (5%) -F_4 (5%) -TGA (5%).

EXAMINATION OF CARDIO VASCULAR SYSTEM

Inspection: nutritional status, RR, recessions, cyanosis (central or peripherall), pallor, clubbing of fingers, dysmorphism (top 3 syndromes: Down's Williams, Digeorge or Turner's , Noonan's), visible pulsations (hyperdynamic apex beat), chest wall deformity.

Palpation

- Apex precordium: thrills (like stroking a cat), turbulence, heaves heart.
- Femoral pulse: if we feel the femoral pulses does this R/O CoA.
- Liver: >2 cm BLCM.

Auscultation

Heart sounds (1 & 2), murmur (systolic or diastolic), murmur intensity (loud or soft) & where is it loudest ?

Innocent or Pathological murmurs

Innocent murmur: the diastolic murmur is never innocent. Innocent murmur is present in at least 50% of normal children.

Still`s murmur: low pitched, vibratory, systolic ejection, ⇧ ē supine position.

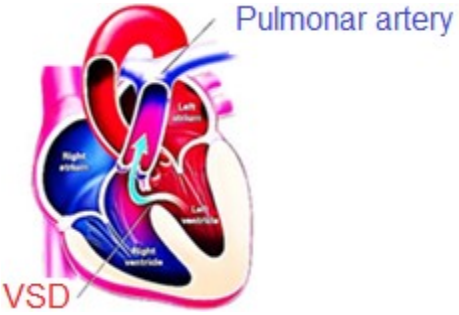
Venous Hum: continuous murmur in supraclavicular region ⇩ on lying down or ē pressure on neck.

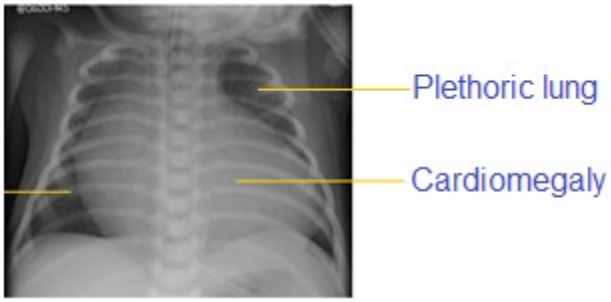
NB: # A baby ē PDA & high pulmonary pressure may have a completely normal examination in the first few wks of life even if there is significant problem ē the heart.

50% of babies ē CHD has no murmur on examination & absence of a murmur does exclude the presence of potentially serious heart disease.

ACYANOTIC CONGENITAL HEART DISEASES

VENTRICULAR SEPTAL DEFECT





VSD is communication between the 2 ventricles, if the defect is small it may passed unnoticed & closed spontaneously in the first few months of life, or even after decades of life, but 15% of cases become clinically symptomatic ē the presence of moderate to large VSD (>5mm in size) & presented ē CHF in the 3^{rd}-4^{th} wk of life. VSD is considered one of the most common causes of CHF after the NN period. In VSD, the O_2 content in Rt ventricle is greater than in Rt atrium.

Incidence: 20% of CHD. Isolated VSD seen in 2-6 /1000 Live births.

Clinical picture

●Dyspnea. ●Tachypnea. ●Difficult breathing. ●Failure to gain weight. ●Sweating while feeding. ●Frequent respiratory infection. ●Pansystolic (holsystolic) murmur along lower sternal border. ●Palpable systolic thrill along left sternal border ●Displaced apex beat as the heart enlarges.

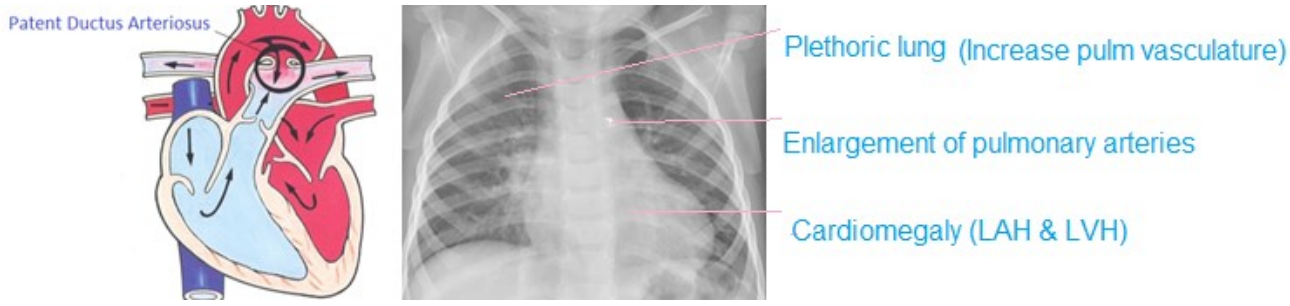
Investigations

●ECG: Left atrial hypertrophy; broad notched P wave in lead II & peak duration ≥ 0.04 seconds, terminal P negativity in lead V_1 ē duration ≥ 0.04 seconds & depth ≥ 1mm. Left ventricular hypertrophy; tall R in V_5 ,V_6 > 25mm & strain pattern V_5 , V_6 ē depressed ST segment inverted T wave in severe hypertrophy, deep S in V_1, V_2, ē or ēout left axis deviation, or biventricular hypertrophy.

●Chest X ray: cardiomegaly & plethoric lung.

●ECHO: localize the size of VSD, cardiac dilatation, evaluation of left ventricular function, detection of presence or absence of other associated defect in the heart.

PATENT DUCTUS ARTERIOSUS



PDA is persistent communication between the descending thoracic aorta & the pulmonary artery that results from failure of normal physiological closure of the fetal ductus. PDA undergoes fairly rapid initial constriction during the first hrs after delivery.

The final functional closure over 1-8 days. In PT infants closure may be delayed up until the time of full gestational age & beyond, a widely PDA is an important & fairly frequent cause of serious illness in the neonate, symptoms of heart failure, growth retardation & may prone to lower respiratory tract infection. The frequency of PDA inversely proportional to advancing gestational age. PDA is present from birth but may not presented until adulthood, when symptoms of endocarditis, pulmonary hypertension, or heart failure may prevail.

Incidence: 1/2000 births. 10% of CHD. The female/male ratio is 2 : 1

Clinical picture

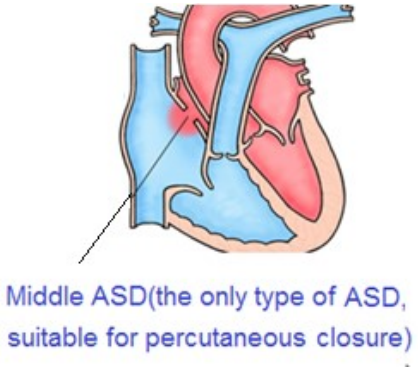
The symptoms depend upon the size of the ductus & how much blood flow it carries, it may cause no symptoms & detected by the heart murmur. The turbulent flow of blood through the PDA puts a person at a higher risk for a serious infection (endocarditis). The symptoms include:-

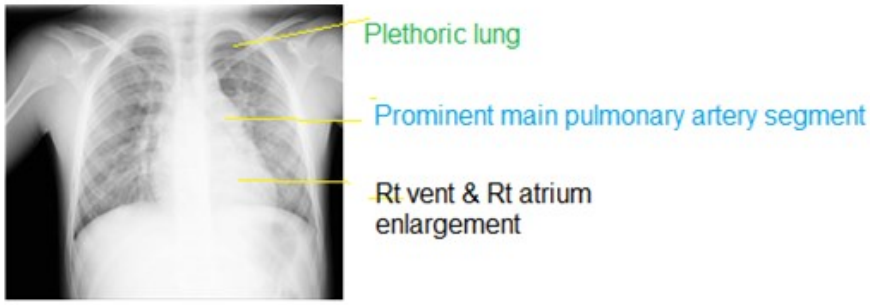
- Poor feeding & impaired growth.
- Sweating while feeding (diaphoresis).
- Dyspnea (shortness of breath).
- Tachypnea (rapid breathing).
- Bounding pulses (peripheral pulse ⇧ in amplitude).
- Widened pulse pressure >25 mmHg.
- Continuous “machinery” murmur, usually heard most clearly at the left upper sternal border & left infraclavicular area are characteristics.
- ⇧ susceptibility to chest infection & endocarditis.

Investigations

- Chest X ray: enlargement of pulmonary arteries & veins, cardiac enlargement (LAH & LVH) & ⇧ pulmonary vasculature (plethoric lung).
- ECG: LAH & LVH
- Echocardiography: confirmatory, demonstration of flow of blood through PDA, it`s size, presence of cardiomegaly & presence of other associated cardiac anomalies.

ATRIAL SEPTAL DEFECT





ASD is a defect in the wall between the 2 upper chambers of the heart. Include 4 typ-es; the commonest (80% of cases) is the ostium secundum, others include, ostium pr-imum, sinus cavernous & coronary sinus ASD. The left to right shunt (because the left atrial pressure is higher than that in right atrium), cause a large volume of blood than normal to be handled by the right side of the heart, this extra blood passes through the pulmonary artery into the lungs causing higher blood flow than normal in lungs.

Incidence: the 3rd most common CHD after VSD & PDA, account for 6-8 % of CHD. Girls have ASD twice as often as boys.

Clinical picture

Isolated ASD in infancy usually asymptomatic & are most often detected at the time of preschool physical examination, sometimes these defects are detected when ech-

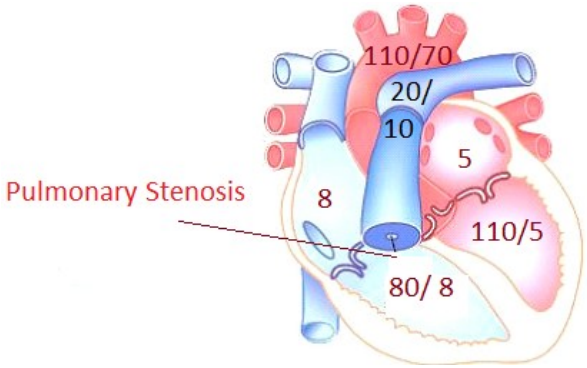
ocardiography are performed for some unrelated reason. A few babies do present ē symptoms of heart failure in infancy, this is uncommon. While ASD is generally well tolerated in infancy & childhood, development of exercise intolerance & arrhythmia in later childhood & adolescency & the risk of pulmonary vascular obstructive disease in adulthood make these defects important. The symptoms include;

- Soft ejection systolic murmur, grade I-II/IV, secondary to ⇧ blood flow across the pulmonary valve, heard best at upper left sternal border.
- Mid diastolic flow rumble (ē the bell of stethoscope), best at the lower left sternal border due to large volume flow across tricuspid valve.
- Fixed splitting of the 2ndHS (fixed, does not vary ē respiration) is the most character-istic sign of ASD).

Investigations

- Chest X ray: varying degrees of enlargement of right ventricle & atrium depending on the size of the shunt, prominent main pulmonary artery segment, plethoric lung (appearance of vessels in the distal lung), cardiac enlargement is often best appreciated on the lateral view because the Rt ventricle protrudes anteriorly as its volume ⇧.
- ECG: shows volume overload pattern on the right ventricle; the QRS axis may be normal or exhibit right axis deviation.
- Echocardiography: detect even small ASD, 100% accuracy, measurement of the size & description of the precise location of ASD.
- Cardiac Catheterization: for detection of O_2 saturation & pressure gradient in different chambers of the heart & in the main blood vessels.

PULMONARY STENOSIS



PS accounts for 7% of CHD, most cases are asymptomatic unless PS is severe. Commonly associated ē Noonan`s sy.(male Turner). Classified into 4 types; Valvular ẃ is the commonest & occur in 85% of cases, Supravalvular, Subvalvular (infundibular) & Branch peripheral PS (affecting either the left or right branch of pulmonary artery).

Mild PS: if the valve area is >1 cm/m^2 & the transvalvular gradient is 30-50 mmHg, or the peak right ventricular systolic pressure < 75 mmHg.

Moderate PS: if valve area is 0.5-1 cm/m^2 & the transvalvular gradient is 50-70mmHg , or right ventricular systolic pressure 75-100 mmHg.

Severe PS: if the valve area is <0.5 cm/m^2 & the right ventricular systolic pressure gradient is >75 mmHg.

In PS the pressure in the Rt ventricle ⇧ while in the pulmonary artery pressure ⇩.

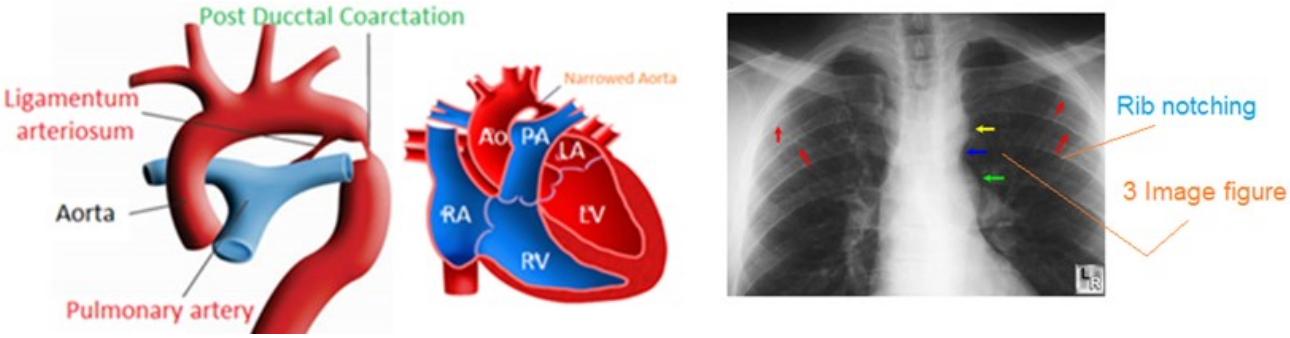
Clinical Picture

•Dyspnoea & tachypnea. •Lethargy & feeding difficulty. •Pale, cool, or clammy skin •Ejection systolic murmur, loudest in the left sternal border (2^{nd}-4^{th} ICS) & radiating toward the left shoulder. •Ejection click often precede the systolic murmur. •Wide splitting of the 2^{nd} HS as a result of delay in right ventricular ejection. •Inaudible pulmonary closure sound (P_2) in severe PS. •There may be a thrill, best felt when the pt leans forward & breathes out. •Prominent α wave in the jugular pulse.

Investigations

- ECG: evidences of RVH : tall R wave in V_1 equal to or larger than S wave in that lead (reversed R/S ratio), deep S in V_6, strain pattern in V_1 , V_2 , V_3 ē depressed ST segment, inverted T wave in severe hypertrophy ē or ēout right axis deviation.
- CXR: usually shows a normal heart size. In severe PS there may be ⇧ in heart size & dilatation "post stenotic" of the main pulmonary artery.
- ECHO cardiography: define where the stenosis lies & how severe it is, evaluation of right ventricular function, systolic pressure & transvalvular gradient.

COARCTATION OF AORTA



CoA is a congenital narrowing of upper descending thoracic aorta, the heart must work harder to keep the blood flowing passed the narrowed area, ẃ may be predu- ctal, ductal, or postductal, most commonly at the site of insertion of the ductus arte- riosus, additional cardiac abnormalities are common including bicuspid aortic valve ẃ occur in 80% of cases, VSD in 40% of cases & ASD, or TGA. The risk ⇧ ē Turner, Sturge Weber, William & Di-George syndromes. The narrowed aorta produces ⇧ in left ventricular overload, wall stress, LVH & CHF.

Incidence

6% of infants ē CHD & the 5th most common cause of CHD in infants. Is 2-5 times more frequently in males than females.

Pathophysiology

Narrowed aorta produces ⇧ Lf ventricular afterload, wall stress, LVH & CHF. The associated pathology include:-

1. Collateral circulation

*Inflow primary from branches of both subclavian arteries (internal mammary A, vertebral A, costocervical, thyrocervical trunks).

*Outflow: into descending aorta, two pairs of intercostal arteries.

2. Aneurysm formation of intercostal arteries: 3^{rd}&4^{th} rib notching (rare before age 10 yr)

3. Coronary artery dilatation & tortuosity: due to LVH.

4. Aortic valve: bicuspid aortic valve (27-45%) & aortic stenosis (6-7%).

5. Intracranial aneurysm: Berry type intracranial aneurysm in some pts.

6. Associated cardiac anomaly: seen in 85 % of cases ē CoA.

Clinical Picture

Symptoms depend on how much blood can flow through the aorta, the presence of other heart defects may also play a role, around 50% of newborn ē this problem will have symptoms in the first few days of life, in milder cases, symptoms may not develop until the child have reached adolescence, symptoms include:-

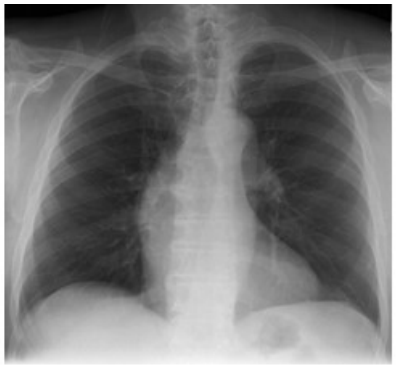
•Dyspnea. •Tachypnea. •Sweating. •Cold feet & legs. •Easy fatigability & poor feeding. •Failure to thrive, poor growth. •Nose bleeding. •Leg cramps. •Dizziness or fainting. •Headache. •High BP in the arms & occasionally the left arm pressure is lower than the right arm pressure if the origin of the left subclavian artery is involved in the coarctation. •BP difference between arms & legs (>20 mmHg). •Weak or delayed pulse in the legs. •Systolic murmur (harsh) or abnormal whooshing sound caused by turbulent blood flow, heard best in the left infraclavicular area & under the left scapula.

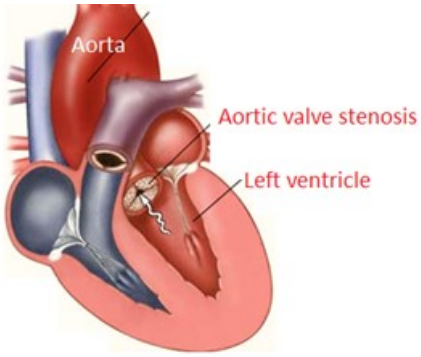
Unfortunately in infants <6 months of age ē CoA & cardiac failure, the diagnosis can be mistaken for sepsis or pulmonary disease in almost 50% of cases.

Investigations

- ECG: may be normal, or show LVH, Left atrium abnormality, 1st degree heart block, complete or incomplete RBBB or biventricular hypertrophy.
- Chest X ray: cardiomegaly, narrowing in the aorta at the site of the coarctation, pos-tstenotic dilatation results in the "3" image is often seen, notching (grooves) on the ribs, commonly seen after 5 yrs of age, results from erosions in the ribs 2ry to tortuous pulsating intercostal arteries. Pulmonary edema if associated ē HF.
- Echocardiography: localize the site & severity of the coarctation, flow. Color doppler measure the peak pressure gradient across the obstruction & left ventricular dimensions & function are assessed by M-mode & detection of other heart defect such as a bicuspid aortic valve ẃ occur in 80% of cases.

AORTIC STENOSIS





Chest x-ray shows prominent of the right mediastinal border occupied by the ascending aorta. The descending aorta is unfolded but of normal calibre. Heart size is normal. No lung or pleural abnormality

AS include supravalvular, subvalvular &valvular types, the aortic valve has 3 flaps, called "cusps" or "leaflets" that open & close during systole & diastole. Baby ē mild AS shows no symptoms & those ē moderate & severe AS can experience dyspnea, tachypnea.

Older children & adults experience dizziness, fainting attacks & easy fatigability, exertional dyspnea, angina & syncopal attacks. The left ventricle initially compensate for the ⇧ resistance caused by AS by thickening to help to eject blood through the stenotic aortic valve. Isolated AS rarely become symptomatic until the aortic valve area is <1 cm & the mean gradient is >40 mmHg or the aortic jet velocity is >4 m/second. Supra valvular AS commonly seen in William`s syndrome.

Incidence: 5 % of CHD & 4 times more likely to occur in boys than girls.

Clinical Picture

- Tachypnea. Dyspnea.
- Sweating while feeding.
- Liver enlargement.
- Palpable left ventricular heave or thrill.
- Pulmonary rales.
- Absent or ⇩ 2nd HS & narrow pulse pressure. .
- Systolic murmur is loudest over the 2nd right ICS & suprasternal notch ē or ēout thrill, transmitted to neck & apex.
- Aortic ejection click.
- Gallop rhythm.

Investigations

- ECG: is frequently normal but may show LVH.
- CXR: slight LVH, plethoric lung..
- Echocardiography: confirmatory, degree of valve obstruction, evaluation left ventricular function & filling pressure, transvalvular gradient, detection of coexisting abnormalities of other valves.

CYANOTIC CONGENITAL HEART DISEASES

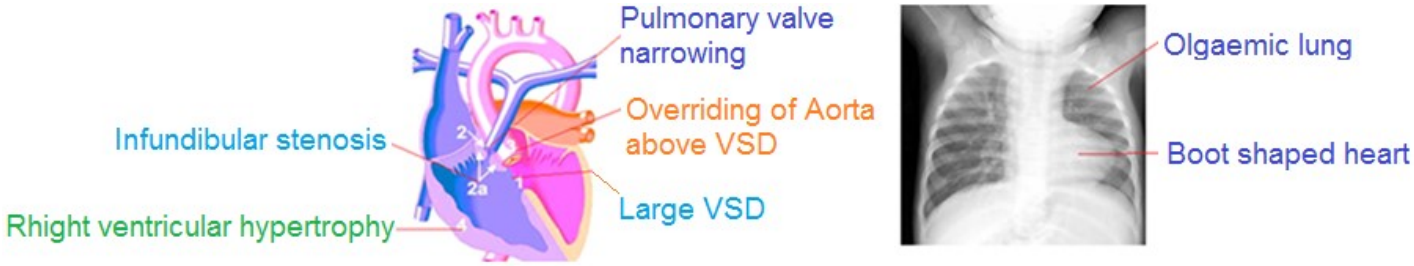
Cyanosis: is it of cardiac or pulmonary in origin?

Hyperoxia test: neonates ē CHD usually do not have significantly ⇧ of PaO_2 during administration of 100% oxygen.

- The O_2 saturation in cyanotic heart diseases is <90% (pulse oximeter) & PaO_2 <60.
- The degree of cyanosis; depend upon the amount of pulmonary blood flow.

The 5 T_s are the most common cyanotic CHD: T4, TGA, TA, Ta, & TAPVR.

TETRALOGY OF FALLOT



Incidence: 2-3/10.000 live births. The most common CHD beyond infancy.

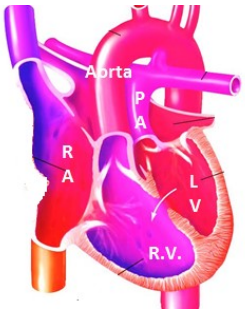
Clinical picture

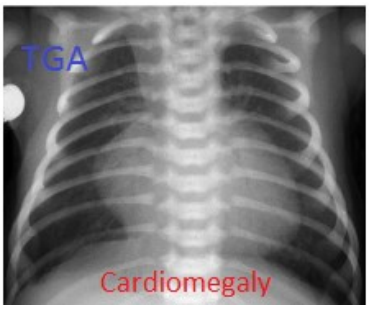
- Central cyanosis & Cyanotic spills.
- Clubbing seen from 3-6 months of age.
- Harsh ejection systolic murmur over the pulmonary area & LSB.
- Thrill along LSB.

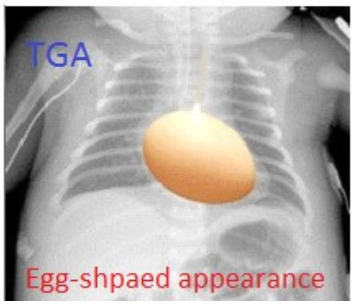
Diagnosis

- CXR: boot shaped heart & oligemic lung.
- ECHO: for anatomy of great vessels, overriding of aorta, PS, RVH & VSD.
- ECG: RVH ē/ēout right axis deviation.

TRANSPOSITION OF GREAT ARTERIES







TGA account for 5% of CHD, the aorta leaves the right ventricle (rather than the left as in normal heart) & takes unoxygenated blood to the body, while the pulmonary artery leaves the left ventricle & take oxygenated blood to the lung (the position of pulmonary artery & aorta are reversed), so that most of the blood returning from the lungs return to the lungs again “lung-heart-lung” & most of blood returning from the body return to the body again, “body-heart-body” ēout being routed to the lungs for oxygen. Infants born ē TGA survive only if they have one or more connections that let oxygen rich blood reach the body, either through ASD, VSD, or PDA. The TGA require surgery, usually in the first wk of life.

Clinical picture

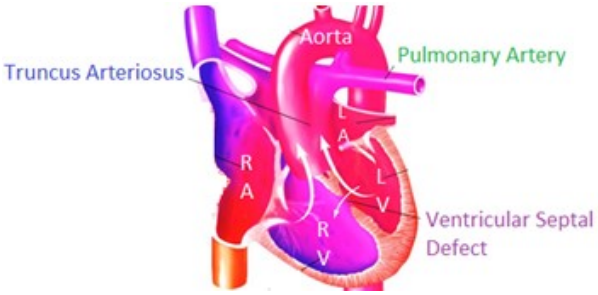
- Cyanosis: if the infant has an intact ventricular septum, cyanosis at birth (at least by 48 hrs because by then the ductus arteriosus has closed), all babies has a PDA at birth that may allow enough mixing to prevent severe cyanosis initially, but as the ductus arteriosus closes, as it typically will in the first hours or days of life, cyanosis become more severe. If infant has a large VSD, less severe cyanosis will be noticed & associated ē CHF.
- Tachypnea: in response to the low O_2 levels.
- Silent heart: no murmur, or are not typical nor always present unless other lesion present e.g. VSD.
- Palpable right ventricular impulse.

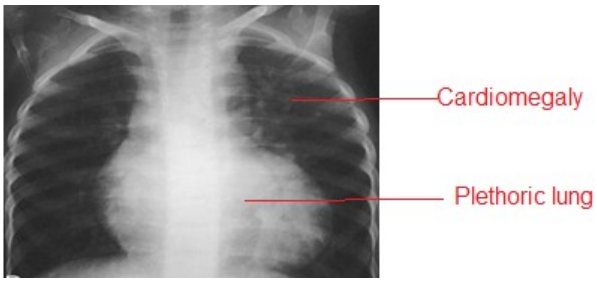
- Engorged neck veins.
- Enlarged liver become apparent in the neonatal period. If untreated, over 50% of infants ē TGA will die in the first month of life & 90% in the first yr. Babies will often develop signs & symptoms of CHF over the course of the first wks or months of life.

Diagnosis

- ECG: may show, RVH, Rt axis deviation & may show myocardial damage due to ischemia
- CXR: narrowed superior mediastinum gives to the cardiac silhouette characteristic egg-shaped appearance, cardiomegaly ē ⇧ pulmonary vascular markings may be found if VSD is present.
- ECHO: anatomy of vessels, presence of other associated anomaly e.g. VSD, ASD, or PDA, are easily seen.

TRUNCUS ARTERIOSUS





TA seen in 2-4% of severely sick neonates ē CHD. The pulmonary & aortic arteries are co-mbined, only one artery arises from the heart & that this artery “TA” gives rise to the coronary arteries, pulmonary artery & aorta, result in ⇧ of blood flow into the lungs & it is always override a large VSD.

Clinical picture

•Cyanosis. •Dyspnea. •Tachypnea. •Excessive sweating. •Pansystolic murmur (VSD), at the left sternal border. •Cardiomegaly.

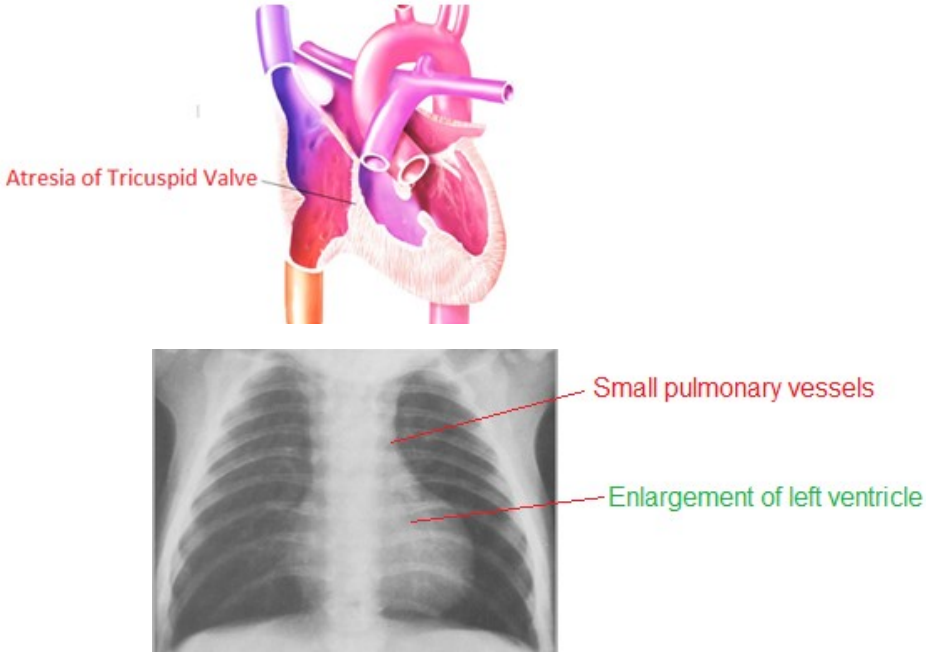
Investigations

•CXR: cardiomegaly, plethoric lung, the combination of right sided aortic arch, strongly suggests TA, however further confirmatory investigations are always needed.

•ECG: RVH & right axis deviation.

•ECHO: anatomy of great vessels, truncal valve, aortic arch & VSD are easily seen.

TRICUSPID ATRESIA



Ta is a congenital agenesis or absence of the tricuspid valve, during the first 8 wks of gestation, resulting in no direct communication between the right atrium & right ventricle, missing tricuspid valve result in an undersized or absent right ventricle & well have ⇩ blood flow into the lung. The blood that return from the body to the right atrium cannot directly enter the right ventricle & most pass through the hole of ASD into the left atrium & then to the left ventricle. An opening may be present in atrial or ventricular level, also

a PDA allow blood to pass through from the aorta to the pul-monary artery & receive O_2 from the lungs. 70% of cases have normal relationship of great vessels & 30% have transposition of great arteries.

Incidence: 5/100.000 Live births & the 3rd most common form of CHD.

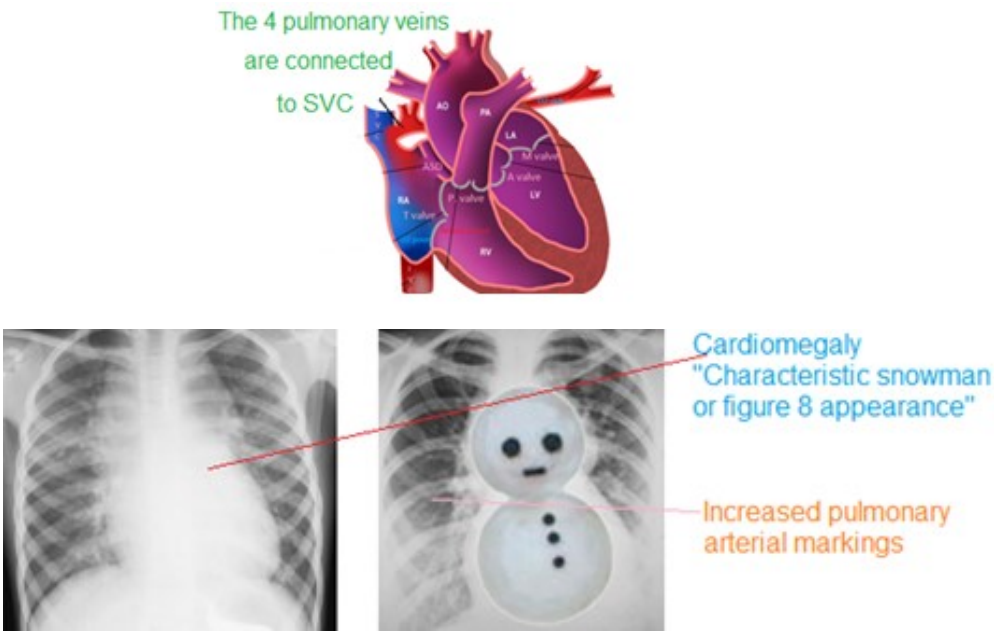
Clinical picture

Nearly 50% of babies ē Ta present ē symptoms on the day of birth & 80% will have symptoms by the end of the first month of life, the magnitudes of pulmonary blood flow determine the timing of & type of clinical presentation. Infants ē pulmonary oligemic exhibit cyanosis in the first few days of life, tachypnea & acidosis, hypoxic spells are not common in the neonate although the spells can occur later in infancy, infants ē pulm-onary plethora usually present ē signs of heart failure within the first few wks of life; dyspnea, fatigue, difficult to feed & a holosystolic murmur on LSB is suggestive of VSD, or soft ejection systolic murmur & splitting of the 2nd HS ẃ is characteristic of ASD.

Investigations

- Chest X ray: enlargement of Left ventricle, a concave left border & small pulm. ves- sels, the aorta is continuous ē the cardiac shadow in left anterior oblique views.
- ECG: LVH & left axis deviation (the only cyanotic type of CHD ē this finding).
- ECHO: confirm the presence of Ta & VSD.
- Cardiac catheterization: is an invasive procedure that gives very detailed information about the structures inside the heart, done under sedation, a small, thin, flexible tube (catheter) is inserted into a blood vessel in the groin & guided to the inside of the heart, BP & O_2 measurements are taken in the 4 chambers of the heart, as well as the pulm artery & aorta & contrast dye may be injected for more visualization.

TOTAL ANOMALIES PULMONARY VENOUS RETURN



TAPVR, the pulmonary veins have no connection ē the left atrium, they drain directly or indirectly into the right atrium. There is total mixing of the systemic venous blood & the pulmonary venous blood within the heart. The systemic circulation dependent on shunting through ASD or patent foramen ovale.

Clinical Picture

•Cyanosis of skin, lips, or nails. •Tachypnea & Feeding difficulties. •Not growing as fast as normal. •Frequent respiratory infection. •Some children does not start having symptoms until later in infancy.

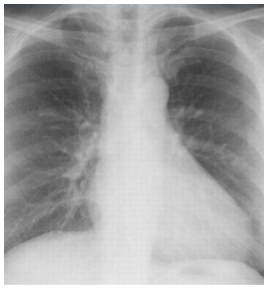
Investigations

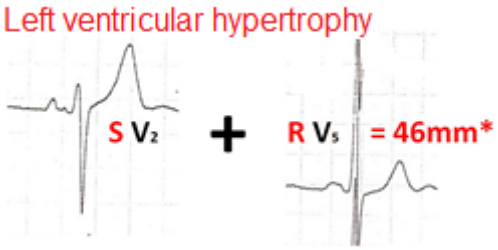
•Chest X ray: there is cardiomegaly with increased pulmonary arterial markings. There is dilation of both the Lf & Rt innominate veins & the right superior vena cava producing the classical "snowman" or "figure of 8" appearance. The superior mediastinum is enlarged secondary to dilation of the right vena cava, innominate vein & ascending vertical vein

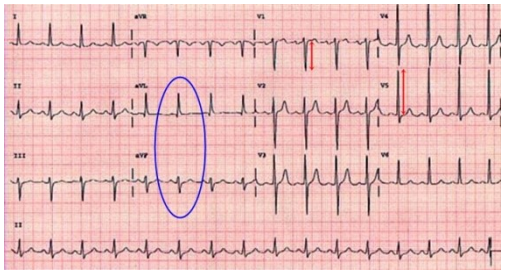
•ECG: shows RVH.

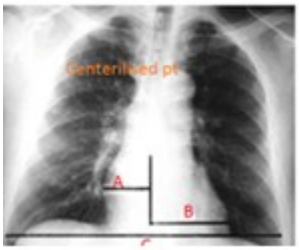
•ECHO: RVH, ASD, or patent foramen ovale ē left to right shunt.

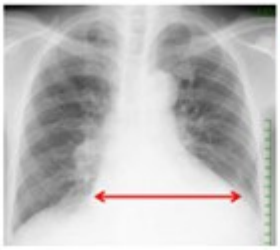
HEART FAILURE

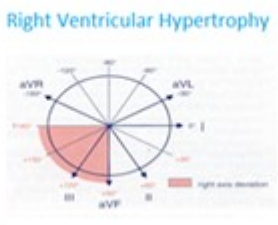


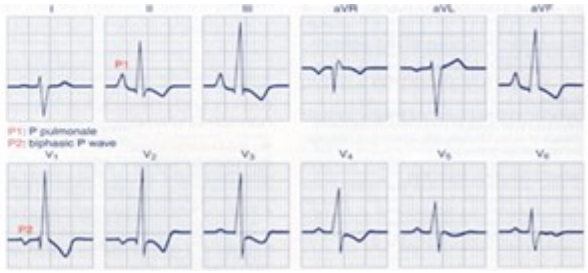












CHF refers to clinical state of systemic & pulmonary congestion resulting from inability of the heart to pump as much blood as required for the adequate metabolism of the body, the clinical picture of CHF result from combination of relatively ⇩ COP & compensatory responses to ⇧ it.

Clinical picture

- Feeding difficulties: important clue in detecting CHF in infants, interrupted feeding (suck-rest-suck cycles), inability to finish the feed, forehead sweating during feeds due to activation of sympathetic nervous system (a very useful sign).
- Tachypnea: > 60/min from age 0-2 months, & > 50/min in 2 months to 1 yr of age & > 40/min in 1-5 yrs of age, it is happy tachypnea ēout much retraction. Grunting (form of +ve end expiratory pressure). Fever ē pulmonary infection may produce tachypnea & in cyanotic CHD, tachypnea may be due to associated brain anoxia & not CHF, the Rx for the 2 conditions is entirely different.
- Tachycardia: > 160/min. in infants, > 100 /minute in old child. Tachycardia ē absence of fever or crying & accompanied by rapid RR & hepatosplenomegaly is indicative of heart

failure. Consider supra ventricular tachycardia if heart rate >220/min. in infants & >180/ minute in older child.

- Cardiomegaly: consistent sign of impaired cardiac function, secondary to ventricular dilatation &/or hypertrophy, may be absent in early stages especially ē myocarditis, arrhythmia, restrictive disorders & pulmonary venous obstruction.
- Hepatosplenomegaly: lower edge of the liver is palpable >2 cm below right costal margin, may be associated ē mild elevation in the bilirubin level & LFTs changes. The ⇩ in liver size after initiation of Rx is an excellent criterion of response to Rx. Usually in such circumstances the spleen is palpable.
- Jugular venous pulsation: seen only in older children & adolescents.
- Pulmonary rales: of not much use in detecting CHF in infants, rales may be heard at both lung bases, when present, are difficult to differentiate from those due to the pulmonary infection ẃ frequently accompanies heart failure.
- Peripheral edema: is a very late sign of failure in infants & children. Facial edema is most common in infants & children, while presacral & posterior chest wall edema in young infants, it indicates a very severe degree of failure. Daily weight monitoring is useful, rapid ⇧ in Wt >30 gm/day in neonate may be a clue to CHF & also useful in monitoring response to Rx. Cold extremity, ⇩ BP, skin mottling, all are signs of impending shock
- Pulse: either pulsus alternans (strong & weak contractions of failing myocardium), pulsus paradoxus (⇩ of pulse volume & BP ē inspiration) are frequently observed in infants ē severe CHF.
- Apical Pulse: visible, diffuse apical pulsation in RVH. Visible, localized apical in LVH.

Investigation

- CXR: look for heart size, contour, pulmonary vasculature, presence/absence of pleural

effusion. In RVH, an angle seen between apex & diaphragm, while in LVH no angle. The earliest sign of heart failure will be cardiomegaly (before pulm. edema). Cardiomegaly is the ⇧ of cardiac shadow > 50% of chest diameter as shown in the diagram below. The cardiac shadow calculated as; horizontal line from must concave point to mid vertical line of centralised pt (a) + horizontal line from must convex point to mid vertical line of centralised pt (b), it is equal to sum of (a) + (b).

- ECHO: the 1st sign of heart failure on ECHO will be enlargement of the filling chambers (left atrium for left sided heart failure, right atrium for right sided heart failure), &/or ⇩ of ventricular contractility.
- ECG: RVH; R wave in V_1, V_2, V_3 > 25 mm, right axis deviation. LVH; R wave in V_5,V_6 > 25 mm, deep S in V_1,V_2, inverted T & left axis deviation
- Electrolytes & CBC: including Ca^+, Mg^+, K^+. & CBC. This helps us to R/O the presence of anemia & electrolyte disturbances. Baby often have mild hyponatremia resulting from ⇧ renal water retention rather than a true -ve sodium balance, mild hyponatremia, ther-efor, does not need to be treated, & administering supplemental sodium may actually worsen the baby`s fluid retention & heart failure. Ca^+ should be administer when hypoca-lcaemia is documented (Ca^+ gluconate 10% 1 ml = 100mg/Kg IV), same as for Mg^+ (Mg^+ SO_4 50% 25 mg/Kg IV). K^+ is important especially when we start to use furosemide, hypo-kalemia may cause cardiac arrhythmia, cardiac arrest, polyuria, paralytic ileus & muscle weakness (dose of K^+cl^- 20% is 2 ml/Kg).

Management

- Diuretics: Furosemide (Lasix), 0.5-1 mg/Kg/day, dose can be ⇧ to 3 mg/Kg/day in severe CHF.
- Inotropic: Dopamine infusion 5-10 mcg/Kg/hr.

- Correction of acidosis: through administration of fluid &/or $NaHco_3$.
- Digoxin: Digitalis Glycoside. digitalizing dose; PO 8-10 mcg/Kg/day, or IV 80 % of the oral dose, maintenance dose is approximately ¼ of total daily dose divided bid, its half-life is 36 hrs, so given once or in two divided doses daily, it is well absorbed through GIT, initial effect can be seen within 30 min. after oral administration & within 15 min after IV administration, adjust the dose in pt ē renal failure. Give ½ the total digitalizing dose immediately & the succeeding 2 quarter doses at 12 hrs intervals, later ECG monitoring. The dose of digoxin is almost never increased but may be decreased in the presence of toxicity or renal failure. Signs of cardiac toxicity include; arrhythmia, bradycardia, AV block & PVCs as premature QRS complex of abnormal shape & duration. Hypokalemia & hypercalcaemia ⇧ toxicity of digoxin &discontinue digoxin if any new rhythm disturbance noted.